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ORIGIN OF HUMAN FACULTY.

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HAVING been requested by the Council of the Neurological Society to read a paper on a recently published book of my own, for the purpose of raising a discussion on the psychological doctrines which are therein presented, I will begin by briefly stating the aim and scope of the book in question.

The title of the book is "Mental Evolution in Man;" but as the work constitutes only the first member of a series which I intend to devote to this topic, its second or subsidiary title more accurately defines the limits of its subject-matter —namely, "The Origin of Human Faculty." The aim of this treatise is twofold. First, to meet upon their own ground those various writers—psychological and theological —who maintain that a great exception must be made in the case of the human mind to the otherwise uniform law of continuous evolution; and, secondly, to indicate the probable causes, and thus to trace the probable history, of the transition between the intelligence of the lower animals and the intelligence of man.

It appears to me that before the Neurological Society I may be allowed to adopt the first of these positions without argument, and will, therefore, assume that in some way or another the transition in question has taken place. On the basis of this assumption I shall be free to devote all the time

at my disposal to a consideration of the probable causes, or method, of the transition. For this purpose it is needful to set out with a brief analysis of ideation.

If I look at any particular face now before me, I receive what is called a perception, or a percept, of that face. If I then close my eyes, or turn them away from that face, but still retain the memory of it before what Hamlet calls "the mind's eye," I have what is designated an image or an idea of the face which I had previously perceived. The idea which I should have in this case would be what Locke calls a Simple Idea—that is to say, the idea of a particular object, or the mere memory of a particular percept. But now suppose that before shutting my eyes I had taken a general survey of all the faces at present before me, I should then have what Locke calls a Compound Idea, or the idea of a face in general, as distinguished from my previous simple idea, or the idea of a single face in particular. It is of great importance to note that these compound ideas are created by a fusion of a number of individual percepts, and thus differ from simple ideas in that they are something more than the mere memories of particular percepts. It is needless to say that animals possess compound ideas as well as simple ideas. For instance, a dog has a compound idea of Man, as distinguished from his particular idea of Master. But, lastly, when we come to what Locke calls General or Abstract Ideas, we find, as he says, "that which puts a perfect distinction betwixt man and brutes." Wherein, then, consists the difference between a compound idea and a general idea? It consists, according to the unanimous agreement of nearly all writers, in the idea having been named by a word, or other sign, which is designedly used as the mark or symbol of that idea. For instance, like my dog, I have a compound idea of Man, and a simple idea of some particular man; but, unlike my dog, I can name the one by the general word Man, and the other by the particular word John. A compound idea, when thus named, becomes what is called a conception, or a concept. Now, it will be observed that this conceptual order of ideation differs entirely from the other two orders which we have just been con-

sidering, in that a symbol is substituted for the mental image, so that the symbol may be used instead of the image, whether or not the image is present to the mind—or, indeed, whether or not any equivalent image admits of being formed at all. Consequently, the mind is now enabled to deal with symbols of ideas without requiring to call up the ideas themselves as memories of perceptions. Consequently, also, the mind is thus enabled to quit the sphere of sense and rise to that of what is called abstraction; furnished with the wings of language, human thought can soar far beyond the possibilities of any ideas which could be suggested by merely sensuous experience.

It will be further observed that the psychological condition to thus naming ideas, so as intentionally to treat the names as symbols of the ideas—the psychological condition required for this is the presence of what is called Self-consciousness. Unless an agent is conscious of itself as a mental agent, and of its own ideas as ideas, it is clearly not in a position to bestow upon them names as names. The mind must be able, so to speak, to get outside of itself, in order to contemplate its own states as such, before it can name these states with the conscious intention of using the names as symbols. In other words, the mind must be capable of introspection; and this power of introspection it is that goes to constitute the one and only distinction between the human mind and mind of lower orders, whether we call this distinction the faculty of Self-consciousness, of Abstraction, of Reason, of Logos, or by any of the other terms which are habitually used to signify this unique power of a mind to turn in upon its own self and examine its own ideas.

Thus far psychologists of every school are agreed. But as a great deal of laxity has been displayed by responsible writers in the use of Locke's terms, and, moreover, as his intermediate division of compound ideas has been largely lost sight of, I have devised for this intermediate division what I think are more appropriate terms, viz., *Generic Ideas* or *Recepts*. Adopting, then, these terms, you will note that all ideas admit of being classified under one or other of

three divisions—viz., Simple Ideas, Generic Ideas, and General Ideas; or, more briefly, Percepts, Recepts, and Concepts. Percepts and recepts are common to the lower animals and to man; but concepts belong to man alone. Moreover, while recepts are formed by an automatic fusion of percepts, without any intentional activity on the part of the mind itself, concepts can only be formed by the intentional activity of the mind in the act of naming a percept or a recept, for the purposes of symbolic abstraction. Thus, a recept is passively received into the mind, while a concept is actively conceived by it. For example, observation shows that water-fowl have one recept (or organised body of percepts) answering to water, and another recept answering to land. So has man. But, unlike the fowl, he is able to bestow on each of these recepts a name, and so to raise them both to the level of concepts. Now, in order to do this, he must be able to set his recept before his own mind as an object of his own thought; before he can bestow his conceptual names on these ideas, he must have cognised them as ideas. In virtue of this act of cognition, he has created for himself—and for purposes other than locomotion—a priceless possession; he has formed a concept.

Nevertheless, the concept which he has thus formed is an exceedingly simple one—amounting, in fact, to nothing more than the naming of some among the most habitual of his recepts, "land" and "water." But it belongs to the nature of concepts that, when thus formed, they admit of being intentionally compared and grouped together into higher and higher concepts, which, in virtue of being successively named, become further and further removed from the sphere of sensuous perceptions. Thus there arises a kind of algebra of recepts. Now, it is in this algebra of the imagination that all the higher work of ideation is accomplished; and throughout it depends on the power of a mind to contemplate its own ideas as such.

The difference between a mind which is capable only of receptual ideation, and a mind which is capable, even in the lowest degree, of conceptual ideation, is usually taken to depend on the absence in the one and the presence in the

other of the faculty of Language. Therefore, it is here necessary to say a few words upon this subject.

The faculty of language is, in the largest signification of the term, the faculty of making signs. Now, there is no doubt that the lower animals present the germ of this faculty. A dog will bark significantly before a closed door as a sign to request that it shall be opened; a wise cat will pull one by one's clothes as a sign to come to her kittens if they are in danger; a parrot will depress its head as a sign to be scratched, and so forth. Nay, a parrot will even use verbal signs with a correct appreciation of their meanings, as proper names, substantives, adjectives, and verbs.[1] Where, then, is the difference between this kind of sign-making, which we may call receptual sign-making, and the sign-making which is peculiar to man, and which alone is conceptual sign-making? The difference is broad and deep. It consists in the power which the human mind displays, as already explained, not only of naming its ideas, but of making one idea stand before another as itself an object of thought. In other words, a man is able to think about his own ideas as ideas. Not only, like a parrot, can he name a particular man John (in consequence of having heard that particular man called John, and therefore associating the name with the man), but he is able to think about this name as a name. And similarly, in all other cases, the difference between naming a thing receptually by mere association, and naming a thing conceptually by intentional thought, is all the difference between knowing that thing and knowing that we know it. And the difference on the side of the talking or sign-making agent, is all the difference between an agent that is conscious only, and an agent that is likewise self-conscious. For it is the faculty of self-consciousness which thus enables a mind to set one idea before another as an object of its own thought; by means of this faculty the mind is able, as it were, to stand outside of itself, and so to perceive objectively the ideas which are passing subjectively—and this just as independently as if it were

[1] All such statements on matters of fact, here and elsewhere, rest upon evidence which is furnished in my book.

regarding an external series of dissolving views. How it is that such a state of matters is possible, whereby a mind can thus, as it were, get outside of its own existence, and so regard its own ideas as objective to itself—this is the mystery of all mysteries, the bottomless abyss of personality. But, accepting the fact as a fact, all that we have at present to do is to note the enormous difference which the presence of this fact introduces with reference to the sign-making faculty. For it means that merely conscious or receptual sign-making is sign-making which is not thought about as such; while self-conscious or conceptual sign-making is sign-making that is thought about as such. Consequently, while a parrot can only learn words or phrases which are stereotyped in the frame-work of special associations, man, after having thus learnt his vocabulary, can afterwards use his words and phrases like moveable types, whereby to convey any number of different meanings by changes of their relative positions. Thus there are names and names; names receptual and names conceptual. In short, it is his super-added faculty of self-consciousness that has made man *par excellence* the sign-making animal; and therefore what we have to do to-night is to consider the genesis of this faculty.

First of all, however, I should like to say something more about the sign-making faculty, as this occurs before the rise of self-consciousness—that is to say, in the brute and in the human infant.

I distinguish four grades of the sign-making faculty. First there is what may be called the *indicative stage*. Long before it can speak, the infant will express its simple desires by means of intentionally significant tones and gesture-signs, such as pointing to objects in connection with which it desires something to be done. Here the infant is obviously at the same level of sign-making as the cat which pulls one's dress to signify "come," or the parrot which will depress its head to signifiy its desire to be scratched.

Next we find what I call the *denotative stage* of sign-making. Here names are bestowed receptually, or by special association, upon particular objects, qualities, actions, and states of feeling. This stage occurs in the child when

it is first emerging from infancy, and is psychologically indistinguishable from that which obtains in the talking birds. Denotative names, then, are names which have been learnt by merely receptual association; they do not imply any self-conscious or conceptual thought.

Following upon the denotative stage is what I call the *connotative*. This consists in a receptual extension of the meaning of a name from the thing which was at first denoted by that name, to other things which are seen to resemble it. Thus, for example, as M. Taine has remarked, a young child which has learnt the name *Bow-wow* for a house terrier, will soon extend it to all other dogs, then to pictures of dogs, to images of dogs, to his elder brother when walking on hands and knees, and so on through ever-widening circles of connotative extension. Now I have observed that a parrot will do precisely the same. One of the birds which I kept under observation, used to bark in imitation of a terrier in the same house. Soon the barking became the parrot's denotative name for the terrier, so that the bird would bark whenever it saw the terrier. After a time it ceased to do this, but would always bark when it saw any other dog. Thus the parrot resembled the child of which M. Taine speaks, in that it extended the significance of its name for a particular dog, so as to apply it to any other dog. Here, however, the connotative extension of the name ceased; the bird would not bark at pictures of dogs, no doubt because it was not intelligent enough to perceive the pictorial representations.

Lastly, there is what I call the *denominative stage* of sign-making, or the bestowal of a name consciously known as such. Here we arrive at what I mean by conceptual naming, and therefore this stage of sign-making cannot arise until the mind has attained to self-consciousness. Therefore, also, it only occurs in man, and first appears in the growing child between the second and third years. Then, of course, the child begins to predicate, or to arrange its names in the form of propositions.

Now, in connection with our subject, it is of the highest importance to note, not only that the three first stages of the sign-making faculty are thus common to animals and

human beings, but also that these three first stages advance very much further in the growing child than they ever do in any animal, even before the growing child attains to the fourth, or distinctively human stage. In other words, even while still moving in the purely receptual sphere, the growing child becomes much more intelligent, and much more proficient in the art of making signs, than any animal. Although not yet a self-conscious agent, and therefore not yet having attained to conceptual thought, a child between two and three years of age has already distanced every animal in respect of its purely receptual intelligence. But observe, thus far no difference of kind can be alleged by our opponents, because to allege any difference of kind between one order of receptual intelligence and another, would be to vacate their whole argument. This argument depends on the distinction between ideation as receptual and conceptual —or between an agent that is, and an agent that is not, self-conscious. But a child up to its third year is not a self-conscious agent. This is proved by the fact that it never employs words having any self-conscious implication, and never gives evidence of even in the lowest degree thinking about its own ideas as such. In short, it cannot be disputed that the respects in which the intelligence of a child between two and three years of age distances that of the most intelligent animal, have reference only to a higher advance of receptual ideation; the ideation has not yet become conceptual, and therefore cannot be alleged by our opponents to differ from the ideation of an animal in kind. The higher degree of intelligence which is displayed by a child of this age must therefore be taken to consist in a higher development of receptual intelligence, just in the same way as a dog is more intelligent than a bird. In order to distinguish this higher degree of receptual intelligence, which only occurs in man, and in the growing child immediately precedes the first appearance of conceptual intelligence, I will call it *pre-conceptual* intelligence.

It is of importance to note how far this higher receptual, or pre-conceptual intelligence, can go, and therefore I will briefly consider the kind of language or sign-making (*a*)

which leads up to it, and (*b*) by which it is expressed when attained.

The indicative stage of language in the infant is at first below that of the more intelligent animals. But very soon it becomes equal to that of the most intelligent. The child will then point to objects in connection with which it desires something to be done, in just the same way as a dog will beg before a water-jug, &c. It will pull one's dress in the same way as a cat does to signify "Come;" and, lastly, it will use its voice to make significant—though inarticulate—sounds, after the manner of all the more intelligent of the higher animals. Thus far, then, the child is still moving in the same levels of receptual ideation as the higher animals. But very soon its receptual ideation begins to distance that of even the most intelligent animal: the ideation of the child has therefore entered upon what I call its pre-conceptual phase. From this point onward its gesture signs become correspondingly more and more significant, so that in children who are late in beginning to talk, it may develop into regular pantomime. But now note, it is impossible that as yet there can be any conceptual ideation, because as yet there are no names, and therefore an absence of so much as the condition to the performance of any act of introspective thought.

Thus much, then, for the indicative phase of language in the receptual and pre-conceptual levels of human ideation. Passing on now to the next, or denotative phase (which the indicative phase may largely overlap in children who are late in talking), we find that when a child first begins to use articulate signs, it learns the use of them in just the same way as a parrot does; that is to say, it learns the name of particular objects, qualities, actions, and states, by special association—in other words, receptually. So far, then, as the beginning of the denotative stage of language is concerned, there is no difference at all between the child and the parrot. Neither is there any difference with regard to the beginning of the connotative stage; for, as I have already said, a parrot will extend its denotative name for a particular dog to all other dogs the resemblance of which one to another it is able to

perceive—just in the same way as a young child will extend its name of *bow-wow* from a terrier to a mastiff. And although the bird will not follow the child where the child takes the further step of extending the name from living dogs to pictures of dogs, this is plainly due to the intelligence of the bird not advancing far enough to perceive the resemblance of pictures to the objects which they are intended to represent. Many dogs, however, and certain monkeys are able to do this, and, therefore, if a dog or a monkey were able to articulate, there can be no doubt that the brute would follow the child through this further step in the connotative extension of a name. Indeed when we remember the extraordinary degree in which monkeys are able to understand the meanings of words, as well as the extraordinary propensity which they show in the way of imitating the actions of mankind, there can be no question that if it were not for the anatomical accident of monkeys being unable to articulate, they would follow a child through what would probably seem a surprising distance in the use of denotative names and receptually connotative words. The chimpanzee now at the Zoological Gardens, which I have taught to count as far as five, displays in a perfectly marvellous degree the power of understanding language—so that one can explain to her verbally what one wishes her to do, in just the same way as we explain this to an infant of about eighteen months old. Therefore, if this animal had been able to articulate, there can be no doubt that it would answer us in the same way that a child answers us when first emerging from infancy.

But here we come to an important point in our comparison between the two cases. After a child does emerge from infancy, its receptual intelligence continues to grow; and it continues to grow until it has left far behind the receptual intelligence of any brute. That is to say, between the time that a child first parts company with the brute in the matter of sign-making, up to the time when it first begins to use denominative words, or words which are used with a true conceptual appreciation of their significance, there is an immensely large interval which is filled by advancing stages of receptual development. Before it has attained to even

the earliest dawn of self-consciousness—and therefore before it has attained to the possibility of thinking about names as names, or of ideas as ideas—the child has made a prodigious advance in its receptual intelligence, and therefore in the sign-making whereby this intelligence expresses itself. Now, as already stated, in order to distinguish this large and important territory of ideation, which is occupied by the mind of a child between the time that its receptual intelligence parts company with that of the most intelligent animal, up to the time when it first reaches the truly conceptual or self-conscious intelligence of a human being, I call this intervening territory of ideation by the name pre-conceptual. Pre-conceptual ideation, then, is that order of higher receptual ideation which is not presented by any brute, but which is presented by the growing child between the time that its developing intelligence parts company with that of even the most intelligent animal, up to the time when the dawn of self-consciousness begins to convert this higher receptual ideation into ideation that is truly conceptual.

I will now briefly consider the kind of sign-making which is distinctive of this pre-conceptual stage of ideation. The child has now acquired a large number of denotative words, which it has learnt by special association to regard as significant of certain objects, qualities, actions, and states. Suppose, then, that it sees its little sister crying. Its denotative name for this sister is *Dit:* its denotative name for the action of crying is *Ki*. Now the object and the action which these two names severally denotate happen to occur together before the child's observation; by the mere force of special association, therefore, the child denotates them both simultaneously—that is to say, brings them into *apposition*. This apposition in consciousness of two habitual recepts with their corresponding denotations is thus effected *for* the child by what may be termed "the logic of events:" it is not effected *by* the child in the way of any intentional or self-conscious grouping of its ideas, such as goes to constitute the distinguishing feature of the logic of concepts. Therefore, when on seeing its sister crying, the child says, *Dit Ki*, although in one sense we may say that the child is making a proposi-

tion, in another and a truer sense we must deny that is making a proposition. The proposition is what may be called pre-conceptual, not conceptual: it is of the psychological kind that we might have expected a monkey to make, if a monkey had been able to pronounce denotative names as well as it can understand them. For the proposition is made by an agent which is not yet a self-conscious agent, and therefore cannot possibly have been thought about as a proposition. That is to say, it lacks the very element of conceptual or introspective thought on which our opponents rely as proving a difference of kind between the brute and the man. Therefore, without argumentative suicide, our opponents cannot afford to maintain that a pre-conceptual proposition of this kind is a genuine proposition, in the sense of being a proposition that implies for its construction any of the distinctively human powers of introspective or abstract thought.

Now, it is needless to say that at this age a child is incessantly making these pre-conceptual propositions; and, of course, the important thing to notice about them is that as yet they are not, and cannot possibly be, conceptual propositions. It is not until the child has attained to self-consciousness, and therefore is able, not only to denotate, but to denominate—not only to name, but to think the names, not only to make statements, but to contemplate its statements as such—it is not until the child has taken this further step that it has the peculiar quality of ideation on which our opponents rely for their psychological distinction between the brute and the man. No doubt these pre-conceptual propositions are strongly suggestive of a near approach to true or conceptual propositions: but the point is that as yet they do not present the very feature which it is necessary that they should present, if they are to conform to the distinction of kind between animal and human intelligence which our opponents have endeavoured to institute. They are always evoked by the external logic of events bringing into apposition objects, qualities, &c., the denotative names of which are called up in the child's mind by immediate association—and, therefore, are necessarily called up in apposition. Thus the apposition which here gives to the

two denotative names the outward form of a proposition, is, as I have before said, an apposition which is furnished *to* the child by the external logic of events; not an apposition which is formed *by* the child through any internal operations of introspective thought. So far, therefore, as any question of kind is concerned, it is manifestly impossible for our opponents to argue that these pre-conceptual propositions betoken anything further than the gesture-signs which characterise the earlier stages of a child's intelligence, and which, as we have before seen, serve to connect its growth with the indicative stage of sign-making as this occurs in the lower animals.

The whole issue, then, here becomes resolved into an enquiry touching the subsequent rise of self-consciousness in the child, or the appearance of the psychological condition to a child thinking about its own ideas as ideas—the psychological condition to its thinking about names as names, and therefore the psychological condition to its raising a merely pre-conceptual statement of a fact which it perceives, into a conceptual statement of that fact with an introspective knowledge of it as a fact.

Now, in considering this final stage, or the rise of self-consciousness in the child, it is of importance to note that even the lower animals present some of the earliest psychological conditions to the subsequent appearance of self-consciousness in the more gifted intelligence of man. Thus, in the minds of brutes, as in the minds of men, there is a world of images or recepts; and this image world, even in brutes, displays a certain amount of internal activity, which is not wholly dependent on sensuous associations supplied from without. The phenomena of dreaming, hallucination, home-sickness, pining for absent friends, and so forth, amply demonstrate that in our more intelligent domesticated animals there may be an internal (though unintentional) play of ideation, wherein one image suggests another, this another, and so on, without the need of any immediate associations supplied from present objects of sense. Furthermore, receptual ideation of this kind is not restricted to the images of sense-perception; but is largely concerned with

the mental states of other animals. That is to say, the logic of recepts, even in brutes, is sufficient to enable the mind to establish true analogies between its own subjective states and the corresponding states of other intelligences. Now at this stage of mental evolution, the individual—whether an animal or an infant—so far realises its own individuality as to be informed by the logic of recepts that it is one of a kind, although of course it does not recognise either its own or any other individuality as such.

Nevertheless, there is thus given a rudimentary or nascent form of self-consciousness, which up to the stage that it reaches in a brute or an infant may be termed receptual self-consciousness, while in the more advanced stages, which it presents in young children who have just emerged from infancy and are therefore beginning to talk, it may be termed pre-conceptual self-consciousness. Pre-conceptual self-consciousness, then, is exhibited by all children after they have begun to talk, but before they begin to speak of themselves in the first person, or otherwise to give any evidence of realising their own existence as such. Later on, when true self-consciousness does arise, the child of course is able to do this, and then only is supplied the condition *sine quâ non* to a reflection upon its own ideas—hence to a knowledge of names as names, and so to a statement of truths as true. But long before this stage of true or conceptual self-consciousness is reached—whereby alone is rendered possible true or conceptual predication—the child, in virtue of its pre-conceptual self-consciousness, is able to make known its wants, and otherwise to communicate its ideas, by way of pre-conceptual predication, as I have previously shown. Now, if I had time, I could further show that the pre-conceptual self-consciousness, of which this is the expression, amounts to nothing more than a practical recognition of self as an active and feeling agent, without as yet any introspective recognition of that self as an object of knowledge.

Given, then, this stage of mental evolution, and what follows? The child, like the animal, is supplied by its logic of recepts with a world of images, standing as signs of outward objects; with a practical knowledge of processes

going on in other minds; and with that kind of recognition of self as an active and suffering agent to which allusion has just been made. But, over and above the animal, the child has now at its command a much more improved machinery of sign-making, which, as we have before seen, is due to the higher evolution of its receptual ideation. Now, among the contents of this ideation is a better apprehension of the mental states of other human beings, together with a greatly increased power of denotative utterance, whereby the child is able to name receptually such mental states on the part of others as it thus receptually apprehends. These, therefore, severally receive their appropriate denotations, and so gain clearness and precision as images of the corresponding states experienced by the child itself. "Mama pleased to Dodo," would have no meaning as spoken by a child, unless the child knew from its own feelings what is the state of mind which it thus attributes to another. Hence, we find that at the same age the child will also say, "Dodo pleased to mama." Now, it is evident that we are here approaching the very borders of true or conceptual self-consciousness. The child, no doubt, is still speaking of itself in objective phraseology; but it has advanced so far in the interpretation of his own states of mind as clearly to name them, in the same way as he would name any external objects of sense-perception. Thus, he is enabled to fix these states before his mental vision as things which admit of being denoted by verbal signs, although as yet he has never thought about either the states of mind or his names for them as such, and, therefore, has not yet attained to the faculty of denomination. But the interval between denotation and denomination has now become so narrow that the step from recognising "Dodo" as not only the object, but also the subject of mental changes, is rendered at once easy and inevitable. The mere fact of attaching verbal signs to mental states has the effect of focussing attention upon those states; and when attention is thus focussed habitually, there is supplied the only further condition which is required to enable a mind, through its memory of previous states, to compare its past with its present; and so to reach that apprehension

of continuity among its own states wherein the full introspective consciousness of self consists.

In confirmation of this my general argument, I must now conclude by observing that, although the advance to true self-consciousness from lower grades of mental development is no doubt a very great and important matter, still it is not so great and important in comparison with what this development is afterwards destined to become, as to make us feel that it constitutes any distinction *sui generis*—or even, perhaps, the principal distinction—between the man and the brute. For even when self-consciousness does arise, and has become fairly well developed, the powers of the human mind are still in an almost infantile condition. In other words, the first genesis of true self-consciousness marks a comparatively low level in the evolution of the human mind—as we might expect that it should, if its genesis depends upon, and therefore lies so near to, those precedent conditions in merely animal psychology to which I have assigned it. But, if so, does it not follow that, great as the importance of self-consciousness afterwards proves to be in the development of distinctively human ideation, in itself, or in its first beginning, it does not betoken any very perceptible advance upon those powers of pre-conceptual ideation which it immediately follows? There is thus shown to be even less reason for regarding the first advent of conceptual self-consciousness as marking a psychological difference of kind, than there would be so to regard the advent of those higher powers of conceptual ideation, which subsequently—though as gradually—supervene between early childhood and youth. Yet no one has hitherto ventured to suggest that the intelligence of a child and the intelligence of a youth display a difference of kind.

I have condensed as much of my main argument as I have found to be possible within the limits of a paper. But, of course, it is needless to say that I am very far from having given the whole. In particular, I have omitted all reference to the latter portion of my treatise, which is concerned with the only direct evidence that we have of the earlier stages of mental evolution in the race. Nevertheless,

although isolated and imperfect, this source of evidence is one of immense importance—standing, in fact, to the science of comparative psychology in very much the same relation as palæontology stands to the science of comparative anatomy; since it serves, by a kind of fossil record, to mark a prehistoric development of ideation, which is curiously analogous to the geological record of a prehistoric development of organisation. Moreover, the evidence thus furnished is of special value on account of its wholly independent character; it is throughout perfectly distinct from the psychological analysis on which we have hitherto been engaged. Doubtless you will already have perceived to what it is that I allude: it is to the independent, and I venture to add, the overpowering, witness of Philology. The gradual evolution of articulate language has preserved for us a kind of palæontological record of the gradual evolution of conceptual thought, with the result of showing that in the life-history of the human species, as in the life-history of the individual child, this conceptual thought derived its origin from these pre-conceptual levels of ideation which have already been occupying our attention. Although it is impossible for me now to give even an outline sketch of this argument from philology, I may conclude by quoting the last paragraph of my summary, in order to give you a general idea of the immense assistance which is thus rendered to the theory of evolution in the domain of human psychology.

"Here, then, I bring to a close this brief and imperfect rendering of the 'Witness of Philology.' But brief and imperfect as the rendering is, I am honestly unable to see how it is conceivable that the witness itself could have been more uniform as to its testimony, or more multifarious as to its facts—more consistent, more complete, or more altogethe overwhelming than we have found it to be. In almost every single respect it has corroborated the results of our psychological analysis. It has come forward like a living thing, which, in the very voice of Language itself, directly and circumstantially narrates to us the actual history of a process the consistent steps of which we had previously inferred. It

has told us of a time when as yet mankind were altogether speechless, and able to communicate with one another only by means of gesticulation and grimace. It has to us described the first articulate sounds in the form of sentence-words, without significance apart from the pointings by which they were accompanied. It has revealed the gradual differentiation of such a protoplasmic form of language into 'parts of speech;' and declared that these grammatical structures were originally the offspring of gesture-signs. More particularly, it has shown that in the earliest phases of articulate utterance, pronominal elements, and even predicative words, were used in the impersonal manner which belongs to a hitherto undeveloped form of self-consciousness—primitive man, like a young child, having therefore spoken of his own personality in objective terminology. It has taught us to find in the body of every conceptual term a pre-conceptual core; so that, as the learned and thoughtful Garnett says, '*nihil in oratione quod non prius in sensu*' may now be regarded as an incontrovertible axiom. It has minutely described the whole of that wonderful aftergrowth of articulate utterance, through many lines of divergent evolution, in virtue of which all nations of the earth are now in possession, in one degree or another, of the god-like attributes of reason and of speech. Truly, as Archdeacon Farrar says, 'to the flippant and the ignorant, how ridiculous is the apparent inadequacy of the origin to produce such a result.' But here, as elsewhere, it is the method of evolution to bring to nought the things that are mighty by the things that are of no reputation; and when we feel disposed to boast ourselves in that we alone may claim the Logos, should we not do well to pause and remember in what it was that this our high prerogative arose? "So hat auch keine Sprache ein abstractum, zu dem sie nicht durch Ton und Gefuhl gelangt wäre." To my mind it is simply inconceivable that any stronger proof of mental evolution could be furnished, than is furnished in this one great fact, by the whole warp and woof of the thousand dialects of every pattern which are now spread over the surface of the globe. We cannot speak to each other in

any tongue without declaring the pre-conceptual derivation of our speech; we cannot so much as discuss the 'origin of human faculty' itself, without announcing, in the very medium of our discussion, what that origin has been. It is to Language that my opponents have appealed; by Language they are hopelessly condemned."

A

CONTRIBUTION TO THE PATHOLOGY OF NIGHT: NOCTURNAL PARALYSIS.

BY CH. FÉRÉ.

Médecin de Bicêtre.

THE influence of night upon animals and on man in health and in sickness has attracted the attention of many investigators. It has been attributed to various factors —sleep, cold, hygrometric condition of the atmosphere, darkness, inactivity—each of which, without doubt, plays a certain *rôle*, but this *rôle* is still badly defined.

In discussing this question there are certain physiological conditions of sleep which deserve consideration. Boussingault shewed that a turtle dove burned 255 milligrams of carbon per hour when awake, but that when asleep the consumption fell to 162 milligrams. Scharling says that the amounts of carbon consumed by a man asleep and awake vary as to 1 to 1·237. During the night also the respiratory movements are less energetic and slower (Becquerel) and the rate of pulsation diminishes.[1] One is compelled to admit, however, that the physiological conditions of sleep are very complex and difficult to isolate experimentally.

Darkness of itself exercises a marked influence on the vital functions. Moleschott has shown that the quantity of carbon dioxide exhaled in the dark is to the quantity exhaled in the light as three to five, and that the proportion varies with the intensity of the light. Bidder and Schmidt have shown that among animals in a state of inanition the loss of weight due to the exhalation of carbon dioxide and to transpiration tends to become equal for day and night when the animals are made blind. The influence of white or coloured

light on animal nutrition and development has been demonstrated during the past few years by the works of Moleschott and Fubini,[2] Uskoff,[3] Gysi and Luchsinger,[4] Schenk,[5] Pouchet,[6] Platten,[7] &c. In addition Edwards had already shown the influence of light on the development of batrachian larvas. The relation established by Darwin between the colour of flowers and the fertilisation of plants by certain insects shows in another way the influence of coloured rays upon animals. Nor has this influence been observed by naturalists only, for "a Mr. de B. declared that his manner of conversation with Madame De—— had changed since she had altered the colour of the furniture in the room from blue to crimson" (Chamfort). Moreover an attempt has been made to utilise the action of the various colours in the treatment of mental diseases. I have shown that in certain subjects this excitatory action may make itself apparent by modifications of the muscular force, of the circulation, or of sensibility, the excitatory value diminishing, as a rule, from red to violet.[8] The action of luminous excitation on muscular energy has been confirmed by the experiments of M. Charpentier on the muscles of the eye.[9] The result of the absence of luminous excitation on the energy of muscular efforts had not previously escaped observation altogether, as Reydelet[10] mentions that night marches are the more fatiguing, and recalls an observation of von Humboldt concerning a Countess of Madrid, who lost her voice at sunset and recovered it at dawn. The effects of sensory excitation, and more especially of visual excitation, which I had recorded,[11] have been also confirmed by the experiments of M. Urbantchich.[12] The influence of luminous excitation on the sensibility of the other sense organs is moreover illustrated by a fact of common experience. Many smokers have remarked that in the dark they cannot tell so well, either by taste or smell, if their cigar be lighted or not.

The diminution of sensibility under the influence of darkness had not completely escaped observation. Taillefer [13] noticed that during the night impressions are received more slowly and are more fleeting. It is easy to verify experimentally the retardation of the reaction when the eyes are

closed. Occasionally I have found an increase of time amounting to seven or eight-hundredths of a second in people perfectly healthy. I have observed that with some hysterical people the effect of darkness exhibits itself by a diminution in the volume of the upper limbs—easy to see with a pletismograph—by an increase of electric resistance, by hemato-spectroscopic modifications,[14] by diminution of the amplitude of respiratory movement,[15] &c.

Many of the lower animals unprovided with eyes still manifest an evident sensibility to light. The observations of Tremblay on hydra, of Pouchet on fly larvas, and of Plateau on blind myriapods, leave no doubt on this head. This dermatoptic function evidently exists to a certain degree in man, and with certain subjects it would probably be sufficient to try opportunely, in order to render it apparent. I have seen in many hysterical persons the modifications of the volume of the hand and of the muscular force that one provokes by occlusion of the eyes notably diminished when a large expanse of skin was exposed to the light.

The lowering of external temperature which occurs during the last hours of the night has also a great influence on vital, and more particularly nervous, phenomena. Helmholtz having determined the rapidity of the nervous current in the nerves of a frog at twenty-six metres per second, found that this rapidity may be reduced to fifteen metres when the temperature is lowered to 0°C. The lowering of temperature affects not only the physiological activity but also the nutrition of the nerves after they have been cut. Among frogs, too, segmentation, which requires only fifteen to twenty days in summer, does not appear till the third month in winter; Vulpian and Philipeaux found that the excitability persisted for six months in winter. Heat, on the contrary, favours in general the fulfilment of nervous functions, and also of the psychical ones.[16]

Modifications of the hygrometric conditions of the atmosphere during the night add their own effects to those produced by temperature. Barral has studied to what extent the humidity of the air causes diminution of the quantity of water exhaled.

The electric condition is connected with the hygrometric state of the atmosphere. During the night atmospheric electricity finds in the humidity of the air a good conductor to or from the earth, as the case may be.

All these conditions favour a falling off of nutrition during the night both among animals and plants. Whatever may be the relative importance of these different factors, their collective influence asserts itself by diminution of the intensity of nutrition during the night—a diminution which entails a lowering of temperature in the organism. Von Boerensprung, Gierse, Ladame, Ogle, Jürgensen, &c., have shown that the lowest temperature occurs from four to seven o'clock in the morning. Women, whose normal combustion appears less intense (Hirn), are more sensitive to these cosmic influences, particularly to the lowering of temperature (Gavarret). This influence of the night, and of the absence of excitation, is evidenced by the physiological conditions of hibernating animals. Marshall Hall[17] had already noticed these conditions, and described the effects of the gentlest excitation of the so kindred animals. The falling away of the nutrition among them is so great that Schiff has observed that in a marmot the atrophy of the inferior end of the crural nerve cut five weeks before was not more advanced than the nerve of a dog after five days. In some individuals affected by a great depression of the nervous system one may observe sometimes during the night such functional modifications that one can truly say they are subject to a veritable nightly hibernation. One has attributed to the night a special influence upon certain physiological acts, such as childbirth, which appears in fact more frequent during the night, and also natural death. This influence remains unexplained, but I may add, as far as natural death is concerned, that the tolerably numerous data which I have been able to bring together give no evidence of the influence of the night in this respect.[18]

Although the night has an evident influence on a considerable number of physiological phenomena, it has a still greater one on the phenomena of disease. Sometimes this influence appears determinative, at other times only ex-

aggerative. A large number of painful diseases present nocturnal exacerbations. The usual hours for osteocope pains are well known. Neuralgia, and articular pains frequently intensify during the night. Gouty attacks in particular often occur towards two o'clock in the morning, when the atmospheric temperature is approaching its minimum. Wilks [19] says that it is at this same hour in which nutrition becomes very low that certain ailments of the stomach, hemoptysis, more especially show themselves, and that among epidemics the cholera commences its attack. In fact, it was ascertained at Munich that out of a hundred cases seventy had begun in the night time. The sweats of phthisical patients, which are more of the nature of paralytic exudations than of active secretions, occur by preference during the night towards early morning. A considerable number of cases of dyspnœa become worse during the night. In spasmodic asthma the attacks generally appear during the night, and relief can occasionally be obtained by the action of a bright light. Laennec has recorded a case of this kind in which the attacks were rendered less severe when the lamp was lit. Epileptic fits often increase in frequency towards the morning,[20] but this increase may be variously interpreted.

The influence of the night was well known to the ancients. Homer calls it the ruler of men and gods. Hesiod describes it as bringing forth all the mischief-making spirits of darkness, and makes it the goddess of misfortune; but what had struck them most of all was the influence it exercised over the mental state. It was the great nurse of sorrows—*nutrix maxima curarum.* (Ovid.)

Night plays a considerable *rôle* in the appearance and development of delirium,[21] and this influence in not due to sleep and dreams [22] alone. M. Baillarger remarked long ago [23] that with some insane people lowering of the eyelids was sufficient to provoke optical hallucinations. Allison [24] has recorded certain nocturnal mental troubles occurring exclusively among overworked men of business.

Nocturnal terrors, according to the note of M. Debacker,[25] occur occasionally in over-worked individuals, among whom

they form a sort of delirium. In studying nocturnal terrors it is advantageous to distinguish two orders of facts. These terrors are most frequently the effect of terrifying hallucinations, which arise during sleep, and persist for some time on awaking, sometimes, indeed, till they are dispelled by the appearance of the light. In certain cases, however, nocturnal terror occurs without reference to any fixed mental representation; it then consists of a feeling of pain akin to that of agarophobia, sometimes, indeed, so intense that one might compare it with angina pectoris, and is caused solely by the deprivation of light, occasionally even in the open day in darkness artificially obtained. It is said that Hobbes was incapable of supporting the absence of light. I have already mentioned the case of an individual who, surprised by darkness in going through a tunnel in an unlighted carriage, experienced a sensation of choking and of oppression on the chest, along with a pain so severe that had he not been caught he would have fallen by the door. Another person in the same circumstances fell in a veritable collapse, with incontinence of urine and fæces. Among many neuropathic people the effect of darkness manifests itself only by melancholic and hypochondriacal tendencies which show themselves at nightfall. Besides, it is well known that many invalids experience an increase of their troubles and fancies at this part of the day, and like to ask for more attention: "the night leaves all its powers to grief and only weakens reason." (de Staël.)

Vesanic suicides often occur in the morning, and the influence of nocturnal depression on thoughts of destruction is clearly shown by the fact that they frequently disappear after the patient has partaken of any stimulant whatever, or even of some light food. This circumstance may be cited in passing as a support for the hypothesis of the organic origin of pessimism which I have elsewhere sustained.

The most characteristic troubles, however, relate to the sensory and motor functions. The influence of the night can manifest itself by indispositions which may moreover be connected with another cause. The powerlessness of drunkards, for example, is obviously increased by the night.

In the morning they experience a general lassitude, or are in a paretic state which frequently affects the lower limbs, sometimes also the member which plays the greatest *rôle* in the exercise of their profession. Under the influence of a painful emotion the paralysis may become complete.[26] The morning impotence of drunkards shows itself particularly in the exaggeration of the trembling movement, which diminishes in consequence of new excitations.

The various impotences which can be properly connected with the influence of the night present a combination of ailments both motor and sensory. The first which may have attracted attention is the incontinence of urine. Of this series J. L. Petit recognises three groups according to their cause, viz., the incontinence of children too lazy to get out of bed, the incontinence of those who sleep so soundly that they are not awakened by the sensations of distension, and the incontinence of those who believe they are relieving themselves somewhere. It is but just, however, to recognise with Trousseau that the first cause of incontinence is a neuropathic predisposition,[27] but the classification of J. L. Petit does not the less merit consideration in so far as it takes account both of motor and sensory ailments, the existence of which is incontestable. The indecision and dislike of movement which causes children to lie in bed until the contractibility of the bladder overcomes the resistance of the sphincter muscle, scarcely differs from the indecision and failure of the will that one observes among many neuropathics under the influence of the night. Besides, it is probable that the sphincter muscle, which is so sensible to peripheral excitation and to emotions of every kind, loses some of its tonicity when luminous excitation is wanting, and that all things being equal it does not resist so well simply because of the darkness. This explanation is by no means theoretical, as the experiments of Mosso and Pellacani have shewn that the bladder is sensitive to all kinds of excitation; and, on the other hand, I have been able to show that in man the energy of the sphincter of the anus, which is functionally analogous to the sphincter of the bladder, undergoes notable modifications under the influence of

sensory, and more especially luminous, excitation.[28] At the same time Mosso and Pellacani admit that in darkness the sensation of distension is weakened.

Another nocturnal syndrome, in which sensory troubles combine with motor ones, is well known under the name of hemeralopia or nocturnal blindness. Although this blindness does not, like nocturnal incontinence, occur exclusively among neuropathics afflicted with congenital nervous exhaustibility,[29] it nevertheless prevails among subjects who have undergone great exhaustion from different causes: (1) or general, as the consequence of acute disease (Gubler), in puerperality (Demeulater), in malaria, as the result of bad hygienic conditions in prisons, on board ship, in the army, in houses of education, where epidemics usually spare the better nourished and better lodged, (2) or local, in consequence of fatigue of the eyes in equatorial seas, or in snow fields. Sanson and Sichel hold that hemeralopia induced by these causes is an insensibility consequent upon too strong stimulation, and comparable with the deafness of men who work in the midst of intense noise. One might, however, with more justice compare it to the transitory anæsthesia which succeeds to the sensory paroxysms of epilepsy, and on which A. Hughes Bennett has recently insisted.[30] These forms of anæsthesia by exhaustion are not rare in hysteria, and I have already shown that they may be sometimes induced experimentally by making the patient wear red spectacles for a few minutes only. One may with perfect right connect these phenomena with motor paralysis from exhaustion, of which I have already had occasion to cite some examples,[31] and of which Mr. Suckling has recently discovered a case.[32] Hemeralopia is not only constituted by a periodic anæsthesia, but in a number of cases it is accompanied by sensorial disorders, such as mydriasis, paresis of the pupil, difficulties of accommodation, diplopia and even strabismus. In fits of nocturnal blindness, in fact, there occurs a series of phenomena quite the inverse of those which appear under the influence of a moderate increase of luminous excitation, which determines an abnormal activity

of the special sensibility, and at the same time of the muscular appendages of the eye. One may therefore say that if hemeralopia be a syndrome due to general or local exhaustion, its attacks are produced under the influence of an insufficiency of the physiological excitant.

Paresis for want of physiological excitation occurs frequently among hysterical people under the form of a general depression of all the functions. On awaking, the patient remains in a condition of general torpor, incapable of movement, accompanied by general insensibility, and occasionally by a remarkable coldness of the skin. In many such cases I have ascertained that the contraction of the field of vision, the diminution in the acuteness of sight, and the chromatic sensibility were much more pronounced a short time after the patient got up than they were a few hours later. The result on nervous exhaustibility of the absence of excitation and of the modifications in nutrition which occur during the night, seem to me to be of interest in the interpretation of other phenomena which are much more frequent than one might expect.

Mr. Weir Mitchell[33] has recorded under the name night palsy or nocturnal hemiplegia cases of paralysis, showing itself usually on awaking, sometimes accompanied by a painful numbness and extending sometimes to the two hands, sometimes over the whole side of the body. This occurrence is most frequent among women at the period of the menopause, but occurs also among men.

Mr. Ormerod has described analogous cases, accompanied by swelling of the hands, in which he was particularly struck by the numbness, and the pricking occasionally severe enough to awake the patient. This numbness and paresis usually came on during the night, but might be induced during the day by certain exertions on the part of the patient, such as washing, rubbing or working with the needle. This last circumstance appears to me to be worthy of special note, for it seems to show in cases of "exhaustibility," where there is a question of prolonged exercise, that fatigue may have the same value as the want of

excitation, and the lessening of nutrition which occur during the night. In the cases of Mr. Ormerod[34] which refer for the most part to women at the period of the menopause, the paretic numbness usually disappeared by simple friction.

Mr. Sinkler[35] has also observed this numbness, especially in women at the menopause. He considers that a certain *rôle* is played by overwork, and thinks that the troubles are due to hyperæmia of the nerve trunks or of the spine, favoured by nocturnal decubitus. These patients were cured by galvanisation of the spine, massage, and ergot of rye. Mr. Saundby[36] has published similar cases with painful numbness, which he compares to that which follows compression of a nerve, accompanied by a blue coloration and coldness of the hands. He considers them to be a form of neurasthenia. The most serious cases he has met with were in men suffering from gastric troubles, who were cured by rhubarb and calomel. Replying to the note of Mr. Saundby, Mr. Notley[37] says he has seen the same disorders among women, and has treated them successfully with iron. He attributes them to anæmia. Mr. Moir,[38] on the other hand, has encountered them only in women at the menopause, usually dyspeptic, who were cured by bromide of potassium. Mr. Steavenson[39] has also observed them in women at the critical age, and is inclined, like Weir Mitchell, to admit that hysteria plays a part. Lastly, Mr. A. H. Smith[40] has recently described similar cases, and suggests that the nocturnal lowering of the circulation plays a certain *rôle* in their production.

The works I have just cited show that a paresis with numbness of the extremities exists, which arises under the influence of the night, disappears temporarily under slight excitation and definitely under a tonic, and at the same time quieting treatment. This paresis, which generally appears in exhausted subjects, may also occur without the influence of the night as a consequence of constrained exercise. In such case we have to deal with a special series of complex symptoms, which differs from the increase of pains one observes during the night in certain neuralgic patients,[41] in

certain forms of parœsthesia,[42] and in erythromelalgy.[43] These paretic states appear to be only exaggerations of the phenomena which occur in a normal condition under the influence of the night.

The more transitory and less accented troubles that one observes every day, so to speak, in a great number of neurasthenics, and particularly in hysterical people, form a sort of transition between normal phenomena of life and those paretic states. As Weir Mitchell has fully recognised, hysteria seems to play an important *rôle* in the etiology of these diseases.

The two following cases may be useful as illustrations:—

Case I.—Mrs. V. came to consult me for the first time at the Saltpetrière on the 12th January, 1885. She was accompanied by her mother, who was over sixty years of age, but still very agile, and looked much younger than she really was. The mother had a painful ovarian spot with slight anæsthesia on the left side. Although the menses had ceased nine years previously, she had been subject to migraine, with attacks of melancholia and occasionally convulsive fits. The father, who had been a drunkard and profligate, had quitted the house twenty-five years before, and no one knew what had become of him. A brother of the father had died in prison when undergoing confinement for swindling. Mrs. V. had had two sisters born after her. The one had died of convulsions connected with teething at the age of eighteen months; the other died of convulsions when only six months old.

Mrs. V. had been a precocious child both physically and mentally. She had walked and spoken at a very early age, and had learned very rapidly at school. She has never had convulsions nor tic, but from the age of six has suffered from frequent migraine, followed by vomiting, and during her whole life her sleep has been troubled by nocturnal terrors and nightmare. Menses began when she was twelve. At the age of seventeen she had an attack of chorea in consequence of worry. This lasted three months, and chiefly affected the left side. At nineteen she was married, and had her first child, a boy, when twenty-three. This child died of convulsions on the eighth day. In the following year she was delivered of a child still-born. During her third pregnancy she had anorexia and vomiting, which ceased spontaneously in the fourth month. The child, a girl, was born at the proper time, and though she has suffered occasionally from

convulsions has been otherwise very healthy. She is now seventeen.

Since her chorea Mrs. V. has always enjoyed good health, and has had no distinctive nervous outburst until about three years ago. At that time her husband died, and she suffered severe monetary losses. This induced insomnia, followed by loss of appetite and emaciation. The state of her affairs being improved by dint of hard work her health became pretty good. About two months ago she had a very abundant metrorrhage, the organic cause for which could not be ascertained. It has not occurred again but she has remained pale, and the menstrual flow has been slight but painful. A few days after this mishap she began to feel constrictive pains in the head extending over the whole of the cranium, but predominating in the postero-inferior region, which appeared moreover to be the seat of a constant pressure. From time to time she heard a cracking noise in the back part of the neck, which resounded in the occipital region of the skull. At nightfall she was seized by painful fancies, of ruin for her mother and her daughter, of illness for all her friends; at the same time she was a prey to unusual pusillanimity and indecision. Her sleep was disturbed by dreadful nightmares. She was widely awake towards six in the morning, but was incapable of making any movement. She suffered from distension of the bladder, but could not even think of getting up. Her limbs seemed numb to her and as if wrapped in cotton. She appeared to know the position of her extremities only, and it seemed to her as if the greater part of each limb was awanting, and her hands and feet had been brought up quite close to her body. This sensation is analogous to that experienced by amputated persons, who say they feel only the extremity of the absent limb. She could make no movement whatever unt her mother entered the room. When daylight was admitted a sensation of numbness and pricking gradually appeared in the extremities of the fingers and toes. These sensations, occasionally very painful, preceded the return of ability to move. It was now, as a rule, about eight o'clock, and by this time the patient could get out of bed, maintain herself in a standing position, and make movements of the arms. The complicated movements of the fingers, however, remained almost impossible. She was thus incapable of fastening her clothes or of taking up a pin. When she had moved her arms and been rubbed for a few minutes her fingers became more supple. From the time she awoke until the nearly complete restoration of movement took in general about three hours. One

day when left in the dark till about ten o'clock she was found in the same helpless condition. Movements of the head and neck and of articulation were not affected. On examination no modification of the external aspect of the limbs could be determined. She suffered pain in the region of the left ovary, and had slight anæsthesia on the same side. The contraction of the field of vision was tolerably extensive, and the patient was insensible to violet rays in the left eye. The left iris is of a deeper brown and the pupil not so large as on the other side. Under the influence of bitters, iron, bromide of potassium, and hydrotherapy, combined with static electricity, all these symptoms disappeared in the space of three weeks with the exception of the pain over the ovary and the hemianæsthesia.

Case II.—Mrs. P——, fifty-two years old, does not acknowledge any antecedent hereditary neurotic tendency. Her mother was rheumatic and died of some form of heart disease. Her father died of cancer of the pylorus. Among her relatives there exists a certain number of arthritic symptoms. She herself has never been ill, but from the commencement of menstruation at the age of fifteen she has been subject to very violent migraine, which returns about every fifteen days. These migraines begin by a supra-orbital pain invariably seated on the left. Little by little this pain extends to the frontal region and is accompanied by an exquisite sensibility of the skin—a sensibility so great that the slightest contact even with the hair causes an insupportable burning sensation. When this pain has persisted from about half-an-hour to an hour the patient begins to perceive scintillations which she localises on the left of both fields of vision. These soon give place to a sort of luminous disc, whose borders are indistinct and seem to vibrate rapidly. This disc, situated towards the left, slowly enlarges and at the same time becomes dark in the centre. In a few minutes the disc has assumed the appearance of a toothed saw hollowed out on the half of its internal circumference, or of a half crown of fortification *à la Vauban*. This toothed wheel is the same colour as electric light, but from time to time red and blue points appear. It is in a state of very rapid vibration. In proportion as the wheel opens and enlarges the centre becomes quite dark and obscure. At the end of an hour the toothed wheel has so enlarged itself as to occupy the limits of the field of vision and forthwith disappears. The patient then declares that she can by this time see only the right half of objects placed in front of her, and that when she looks at people they appear to her to be cut exactly in two on

the median line, and she can see nothing of what is on her left; there is in fact hemianopsia, that is to say, loss of the left half of the two fields of vision. Vomiting of food or bile then occurs according to circumstances, but the frontal pain and the hemianopsia persist for several hours and only disappear when the patient is about to fall asleep. After a certain number of attacks the patient has felt for many hours a painful numbness in the left hand and sometimes in the forearm, but never higher. She has never experienced such numbness in the face or in the tongue.

For nearly thirty years this woman has been subject to ophthalmic migraine. Up to the age of the menopause she never complained of any other nervous symptoms. She has had four children, the youngest being fifteen years old and all quite healthy. The menses ceased when she was fifty, without causing any important general disorders. From this time, however, the migraines have undergone a certain modification. As soon as the vomitings have ceased the patient is seized with an irresistible impulse to run away, and it is with great difficulty that one can prevent her by closing with care all the issues of the house. She subsequently recollects this impulse but cannot assign any object in it; she is simply conscious of an invincible desire to walk straight forward. This desire disappears with the pain in the head.

During the six months which followed the menopause Mrs. P—— was greatly overcome by the death of her father and mother, and by the entry of one of her daughters into the convent. Her health greatly changed; the appetite became capricious and at intervals entirely disappeared; her sleep, which had always been good, was disturbed by nightmares; in walking she soon got out of breath; her temperament, which had always been equable and gay, became difficult and altered from sad preoccupations. For nearly a year she has felt a feebleness in her lower limbs, and it is with great repugnance that she attempts to go up a single flight of stairs. For the last six months her condition has been aggravated by the appearance of spots and zones of pain. One of these last, about ten centimetres long by three broad, has arisen between the shoulder blades a little to the left of the median line and corresponding pretty closely with the vertebral groove. The patient complains there of sudden shooting pains, and in addition the skin is sensitive to the slightest touch, while heavy pressure is quite insupportable. Another painful zone of the same nature and about equal size lies in the median line at the bottom of the lumbar region. A third, some-

what rounded in form and about ten centimetres in diameter, occurs under the left clavicle. These painful tracts had existed for more than two months when on the 15th January, 1888, she began to feel an extremely violent pain in the tendo Achillis of the left side. There was no swelling, but pressure was exceedingly painful, as also was the slightest movement of the feet dorsally. The doctor who was called in suspected gonorrhœal rheumatism and proceeded to an examination of the genital organs. These were found to be perfectly sound, but the examination—the true object of which had been concealed from the patient—provoked very strong excitation, followed by a fit of sadness, out of which it was impossible to rouse her. Her appetite completely disappeared, she could take food only in liquid form, and her sleep became more and more troubled. In about a week she began to complain of sudden shocks in the head, which awoke her abruptly. These occurred by night four or five times. Finally came other troubles which raised her inquietude to a climax. When her sleep became broken towards four or five o'clock in the morning she found that she could not move any of her limbs. This general helplessness did not last very long, for after a few minutes of effort she recovered power of movement in her right hand and foot, but for the limbs on the left side prolonged friction was necessary. This paresis was accompanied chiefly on the left side by a sensation of painful numbness and of pricking. The hand especially was quite cold, and the fingers appeared to diminish in volume, the rings hanging quite loosely.

This helplessness, which at first persisted only a few minutes, was in a month's time prolonged for an hour or more. The patient could not raise herself out of bed until someone had opened the windows widely and rubbed her energetically. Even then she would remain for many hours incapable of making any delicate movements or even of simply fastening her dress. When the paralysis was at its maximum the patient declared that she was no longer conscious of the existence of her own body, that she was, to use her own words, a "*pur esprit.*"

When I saw the patient on the 20th of June, 1888, she had been suffering from these paralytic affections for about two months. As it was impossible to put her under a regular discipline in her own house I advised her to enter a hydropathic establishment. Under the influence of cold douches repeated twice a day at regular hours and of a tonic treatment (iron, nux vomica and arsenic) along with bromide of potassium given every

evening in moderate doses (one to three grams) her condition rapidly improved. The paralytic numbness on awaking diminished at once in duration and then in intensity. At the end of fifteen days it had almost completely vanished. The sensation of shocks in the head, which had caused the patient to awake, disappeared in turn. The anorexia and pains resisted longer; at the end of six weeks the pain in the tendon of Achilles still persisted, but in time it too disappeared.

These facts, which I could multiply if necessary, present the greatest analogy with those mentioned by the authors I have already cited. They are perhaps better adapted for showing the *rôle* of neuropathic predisposition and of depressing conditions in the pathogenesis of these nocturnal paralyses. These paralyses grow, so to speak, on the same soil as paralyses by exhaustion, but instead of being determined by an excessive toil, by too intense a sensation,[44] or sensorial[45] excitation, or by the mental representation of one of these conditions of exhaustion[46] they are the result of a *deficiency* of physiological excitation—they are in fact paralyses caused by non-irritation (*paralysies par inirritation*).

Powerlessness by non-irritation can affect all the motor and sensorial functions, and in consequence the intelligence in all its manifestations. But it is quite useless to insist here on the influence of the immediate surroundings on intellectual development.

Experimental studies on the influence of the excitations of one sense on the action of the others leads us to foresee that the detailed study of the special sensibility in these cases of nocturnal paralysis will give interesting results when one can carry it on under favourable conditions. Moreover it is not only the motility or the cutaneous sensibility and the cephalic senses which are affected, but the genital sense as well. For many years I have attended a patient aged thirty-eight belonging to Morel's class of *dégénérés à stigmates physiques et psychiques*, whose impulsions are worthy of special study, and who has been incapable all his life of giving himself up to sensual pleasure otherwise than in open air or in a room lighted *a giorno*. He has, however, never spared the means of supplying the necessary physiological excitation of light.

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THE TREATMENT OF CERTAIN DISEASES OF THE SPINAL CORD BY MEANS OF SUSPENSION.

BY MOTSCHUTKOVSKY, M.D., OF ODESSA.

I FIRST noticed an elongation of the body when witnessing the suspension of patients for surgical purposes in the process of application of Sayre's jackets. Repeated measurements convinced me that this elongation varied from $2\frac{1}{2}$ to 5 cms. and that it involved the spinal column. Two problems occurred to me in consequence of this observation.

(1) What change does take place in the relations between the spinal cord, its membranes and nerve roots, and the vertebral canal during the process of suspension?

(2) Can suspension have a beneficial influence in certain diseases of the spinal cord?

I shall describe the results of my researches in chronological order and begin with the second question.

The history of the first case in which I tried suspension is as follows:—

CASE I.—P. M., from Cherson, aged forty-nine years. At the age of twelve he suffered from sores in the axillary and inguinal regions. These sores (which have left no scars) were cured according to the patient's statement by the use of certain powders. M. is unmarried, has never been addicted to onanism, has not suffered from acquired syphilis, has not exceeded sexually nor in drink. He was for many years in the service of a jobmaster, and on several occasions was severely injured. About twenty-five years ago he fell off a horse for the first time and injured his back. As a consequence he was laid up at this hospital for about six weeks. Five years after the first accident he again fell from a horse upon his back; on this occasion he recovered in about two weeks without any treatment. Finally in 1871 his horses ran away and he fell from the cart dislocating his left ankle. He

was again treated at the Odessa "Town Hospital" and was well in three months. After leaving the hospital he began to suffer from severe shooting pains in the arms, legs, back and waist. These pains he considered to be rheumatic and attributed them to his having taken cold, as he was much exposed to the weather in the course of his work. Up to the year 1880 the pains were severe chiefly immediately before and during bad weather; at other times he felt pretty well. In January 1880 his legs gradually got weak; afterwards his arms were similarly affected. He next noticed that both his arms and his legs were getting thin. The pains now became constant and intolerable, not allowing him to sleep. The patient became unable to walk towards the end of 1881. On admission into the Odessa "Town Hospital" on the 15th of February 1882 patient could stand only with difficulty, and staggered considerably even with his eyes open. As soon as his eyes were closed he fell. He had great difficulty in walking forwards, although supporting himself with the aid of a stick. His feet were raised very much at each step and then rapidly brought to the ground. He could walk backwards much more easily even without a support; however, after a few steps he used to fall on his back. M.'s height was 1·515 metres; he was fairly well built, and weighed 49·65 kilos. The muscles of both upper extremities were somewhat flabby and slightly atrophied about the palms. When the hands were extended the fingers had a bird's-claw appearance. The muscles of the leg, the extensors of the foot especially were also flabby and said to have become wasted. The toes dropped considerably. Dynamometer to right hand, 22 lbs.; left, 20 lbs.; right leg, 85 lbs; left, 82 lbs.

The spinal column was symmetrical and normally sensitive to percussion and pressure. All the joints were normal; mucous membranes vividly coloured; skin flabby, without scars or spots.

On closing his eyes patient could not co-ordinate the movements of his legs. Even under guidance of vision, in an effort to touch some object with the toes his foot was carried considerably beyond the object aimed at. The same phenomenon though in a much less degree was noticeable in the movements of his fingers. Elbow and knee jerks were absent. Temperature normal. Skin always dry, but not scaly. There were several dark brown pigmented spots on the thighs, the marks of slight injuries.

The musculo-cutaneous pain reflexes were examined [by means of a special reflexometer contrived by Dr. Motschutkovsky] on 15th of April and their latent period was determined.

Great toe of right foot, latent period	1·460″
,, left ,, ,, ...	1·738″
Index finger of right hand ,, ...	0·898″
,, left ,, ,, ...	1·0008″

The time requisite for the central conduction of painful impressions (*i.e.*, the time elapsing between a reflex act and the perception of a painful sensation) was as follows:—

Great toe of right foot	0·767″
,, left ,, ...	0·951″
Index finger of right hand	0·371″
,, left ,, ...	0·492″

Sensibility to pain was lessened in the inferior extremities more than in the superior, and on the left side more than on the right. Tactile sensibility was also diminished but not to the same extent, Muscular sensibility, the muscular sense and the sensation of position, were weakened. Thermic sensibility was fairly good, the patient being able to distinguish a difference of 2° in temperatures ranging from 14° to 35° R.

The faradic excitability of the muscles was diminished. The nerves and muscles of the extremities did not respond readily to the galvanic current, though the A and K contractions followed normally in their respective order. The patient complained of formication in his legs, of a feeling of pricking in the ends of the fingers of both hands, of a constant sensation of cold in the legs and of severe shooting pains in the region of the sciatic nerves. The pains were more severe in the right leg than in the left; they were intensified chiefly by sudden barometric changes. Moreover the patient complained of girdle pains about the abdomen and chest and sometimes of pains in the region of the cervical spine, extending into the arms. Sexual functions abolished. Mental state undisturbed. Taste and smell normal, but hearing deficient since fifteen years. Patient could distinguish the ticking of a watch with his right ear at the distance of 8 cms., and of 5 cms. with his left. The tympanic membranes were capped and dull. The pupils were contracted, and reacted only slightly to light. Field of vision normal; acuity diminished. Discs sharply defined, somewhat glistening. Retinal vessels not fully distended. Palpebral conjunctiva vividly injected. Sclerotic vessels dilated.

Chest tympanitic on percussion; expiration prolonged.

Defæcation is normal but micturition infrequent; the patient has to strain before beginning to pass water. The urine yellow, phosphatic, but without albumen or sugar; sp. gr. 1021. The patient was suspended on the 24th of April 1882 for the

first time. After the second suspension the sciatic pains ceased, the sensation of cold in the legs was diminished; stick no longer necessary to stand. After eighteen suspensions the patient with his eyes open walked very well, though with some jerking forwards of the feet. He can stand on one leg for two minutes and walk without a stick with the eyes closed. After the twenty-ninth suspension sensibility to pain was much improved; no more formication and pricking in the tips of the fingers. The sensation of cold has been replaced by a pleasant feeling of warmth. July 1st the musculo-cutaneous pain reflexes also show improvement.

Great toe of right foot, latent period	0·923″
,, left ,, ,, ...	1·102″
Index finger of right hand ,, ...	0·722″
,, ,, left ,, ,, ...	0·790″

The time requisite for the *central* conduction of the sensation of pain was—

For the great toe of right foot	0·449″
,, ,, left ,, ...	0·531″
Index finger of right hand	0·251″
,, ,, left ,, ...	0·370″

Knee jerks still absent. Dynamometer, right hand, 33lbs.; left, 28lbs.; right leg, 90lbs.; left, 86lbs.

By the 14th of November after seventy-seven suspensions the musculo-cutaneous pain reflexes showed:—

Great toe of right foot, latent period	0·741″
,, left ,, ,, ...	0·852″
Index finger of right hand ,, ...	0·369″
,, left ,, ,, ...	0·280″

The time requisite for the central conduction of the sensation of pain was—

For the great toe of right foot	0·320″
,, ,, left ,, ...	0·482″
For the index finger of the right hand	0·204″
,, ,, ,, left ,, ...	0·137″

By that time he had so much improved that, according to his own expression, it was "unnecessary" to feel any better. He walks a good deal about the garden; can walk half a verst (two-thirds of a-mile) without resting and without using a stick. He can go up a flight of steps also without resting. The legs are painless and stronger. There still are some dull girdle sensations and occasional headaches, the latter being in fact more marked since suspension. Knee jerk still absent, but more frequent and

marked returns of sexual feelings. Dynamometer pressure increased: right hand, 58lbs.; left, 46lbs.; right leg, 97½lbs.; left, 89lbs. Weight of patient, 49·4 kilos. On the 15th of March 1883 the following facts were ascertained:—

Musculo-cutaneous "pain-reflexes," latent period—

Great toe of right foot	...	...	0·722"
,, left ,,	...	...	0·795"
Index finger of right hand	...	...	0·232"
,, left ,,	...	...	0·202"

The time requisite for the central conduction of the sensation of pain was—

Great toe of right foot stimulated	...	...	0·320"
,, left ,, ,,	...	...	0·490"
Index finger of right hand ,,	...	...	0·200"
,, left ,, ,,	...	...	0·151"

Dynamometer: right hand, 59lbs.; left, 57lbs.; right leg, 98lbs.; left, 92lbs. The suspensions had to be given up at intervals for a couple of weeks or more, owing to pains in head and spine. They were usually carried out every other day, lasting at the onset for from one to two minutes, later on from four to seven minutes each. After the twelfth suspension an ecchymosis occurred in the left sclerotic lasting about ten days.

The general results of ninety-seven suspensions were:— (1) complete disappearance of shooting pains; (2) diminution of ataxic gait; (3) considerable improvement in the delaying of the musculo-cutaneous "pain reflexes;" and (4) of the conduction of painful impressions; (5) disappearance of certain paræsthesiæ (sensation of cold, of pricking, formication, girdle pains); (6) increase of muscular force in the extremities; (7) improved equilibrium when standing or walking on closing the eyes; (8) improvement of sexual feelings; (9) returning perception of pain in the toes and improvement of tactile sensibility; (10) increased size of atrophied muscles. Throughout the period of six months while suspension was being carried on, no change was noticed in the (1) body weight; (2) knee jerk; (3) pupil and rest of visual apparatus.

Case 2.—N. T., aged forty-two years. Duration of illness, five years. From the age of nine up to the time when he was admitted to the hospital, patient worked in a tobacco factory. Eighteen years ago he had a chancre which healed in the course of a month. It was accompanied by two buboes in the left groin. They were opened and suppurated for about three months. At the same time he had an abscess in the right axilla and a sore

throat; the latter was however not ulcerated. Five years after this a pink rash appeared on his chest, abdomen and limbs. This rash persisted for about three months. The first syphilitic manifestation was treated by means of mercurial pills, Zittman's decoction and iodide of potassium. The eruption disappeared without any treatment; no further signs of syphilis have since been observed. Twenty years ago N. T. gave up drinking, to which he had been addicted for about six years. Married about four years ago and has one child whose health is perfect. Masturbation between fourteen and seventeen years of age; since then venereal excesses especially from 1870 to 1874. He used to live in Roumania where he had no ague, but four attacks of a fever of a typhoid(?) type (at the ages of eight, eleven, sixteen and eighteen years.)

Present State.—Weakness of legs; unable to get up from the bed or to stand for a single minute even with assistance. Micturition frequent; bowels act only every four to five days; complete sexual impotence; severe shooting pains in the arms and legs; abdominal girdle pains; occasional cutting pains about navel, followed by vomiting or diarrhœa (gastric crises); cold creepy sensations in the legs. Patient weighs 51·5 kilos; slightly built and anæmic. Keeps lying on his back, occasionally sitting up for dinner. Bony framework, normal; spine, symmetrical, but sensitive to pressure and to percussion in the cervical region. No roughness of knee joints though the synovial membranes are somewhat full. No true atrophy though muscular system generally flabby. Dynamometer: right leg, 45, left, 43 lbs. Squeeze of right hand 36, left, 24 lbs. On attempting to walk in the normal position no loss of power, but inco-ordination is noticed, the heel striking the ground before the toes; foot lifted with effort and a slight jerk, brought down with a stamp of the heel. Inco-ordination is very marked in movements executed when lying down; when told, his eyes being closed, to touch one knee with the opposite foot, patient raises the latter with two or three sweeps of the leg in the air, and finally misses his aim. Romberg's symptom is well marked; no inco-ordination of arms, no tendon reactions, nor cremasteric and abdominal reflexes. The latent period of musculo-cutaneous "pain-reflexes" are delayed (as measured by my reflexometer):—

Right big toe	0·736″
Left ,, ...	0·699″
Right index finger	0·276″
Left ,, ,, ...	0·325″

Integuments flabby and wrinkled; palms and soles bright red; skin of limbs never sweats and is cold; hair began to fall off about a year ago; nails thick and clubbed. Pin prick distinctly felt over upper trunk, arms, and head. At a lower level, from Poupart's ligament in front and from third lumbar vertebra behind, sensibility to pain is diminished; soles anæsthetic to pin pricks. Examination with Weber's compasses gives similar results. Touch normal except slight alteration in the toes, where the contact of camel's hair brush is felt as a pricking or burning. Sensibility to moderate temperatures chiefly affected; thus 14° C. and 24° C. are not discriminated; between 30° C. and 42° C. differences of 2° to 3° C. are appreciated pretty correctly. The muscular sense is much diminished; muscular sensibility less so.

Neuro-muscular galvanic reactions normal; excitability of calf to Faradic current somewhat diminished. The patient complains of a cold heavy feeling, passing sometimes into formication, in both soles. Severe lightning pains, shooting from buttocks down into great toes. Occasional attacks of perforating pains in the middle third of the legs and in the shoulder joints, especially on the right side. They increase at night, and though unaffected by temperature, they become intolerable during changes from wet to fine weather, and strong N. or N.E. winds. Morphia alone controls these pains. Bowels act but every four or five days; stools scybalous; occasional colics and diarrhœa. Micturition strained and frequent (thirty to forty times daily); sexual sense in abeyance since one year; sleep good in the absence of pains. No optic changes; pupils small, react feebly to shading. The other organs of sense and the mental faculties are normal; memory somewhat impaired. No thoracic nor abdominal symptoms of any importance; quantity of indican in urine.

We diagnosed a sclerosis of the posterior columns of the spinal cord, the patient being in the paralytic stage of the disease. The first suspension was effected on the 12th of May 1882 and lasted two and a half minutes. After this the patient was suspended on alternate days, or at longer intervals, for periods gradually increased to ten minutes. Seventh suspension: patient is able to get out of bed without assistance. Eleventh: begins to walk. Sixteenth: able to sit down and get up from a chair without assistance of hands. Twenty-second: shooting pains begin to subside.

Latent period of musculo-cutaneous "pain-reflexes":—

Right big toe ... ... 0·707"

Left big toe	0·710″
Right index finger	0·280″
Left ,, ,, ...	0·322″

Dynamometer: right hand, 92 lbs.; left, 82 lbs. Right leg, 107 lbs.; left, 98 lbs.

After sixty-one suspensions erections occurred, and micturition was unstrained and less frequent. Bowels act daily. Indican diminishes. The pupils less contracted in the light, dilated more in the dark. Weight, 49 kilos. Perception of painful impressions increased, a slight prick being readily felt. No change in thermic and tactile sensibility. Also the muscles react to faradism. Pain reflexes after sixty-five suspensions:—

Right big toe, latent period	0·712″
Left ,, ,, ...	0·664″
Right index finger, latent period	0·278″
Left ,, ,, ...	0·320″

Summary of progress after eighty suspensions, spread over ten months: (1) No shooting pains; (2) Some locomotion restored, patient able to go quarter of a verst without resting; (3) Micturition normal; (4) Bowels more regular, hence (5) diminished indican; (6) No gastric crises; (7) Return of sexual feeling; (8) Muscular strength of limbs improved; (9) Perception to pain in legs improved; (10) Ataxia less; (11) Pupil action improved; (12) Stands better with closed eyes. The treatment had no effect (1) on the latent period of the musculo-cutaneous pain reflexes; (2) tendon reactions still absent.

Case III.—E. S., aged fifty-five, formerly a soldier. Present illness began about eighteen years ago, the first symptom being an attack of shooting pains in both legs. This came on after exposure for a whole night on sentry duty to a temperature of $-49°$ C., that is $-56°$ Fahr. Since then the pains used to return in bad weather, but were mitigated after a course of brine baths in 1876. Since January 1880 they have again been very intense. No syphilis. He has four children, having married at the age of twenty, and previous history of excess "in venere" though not "in baccho." When in military service he once fell from a cart on to his back and lost consciousness; the seat of injury was painful for a couple of days after. Patient (1) is unable to walk or stand; (2) has severe shooting pains, as well as a cold, creepy sensation in the legs; (3) is blind; (4) suffers from constipation and incontinence of urine; he has also lost sexual power, sleep, and appetite. He is a man of average build, and looks intelligent and truthful. His habitual attitude is lying on

his back, being unable to sit up for more than five minutes at a time or to lie on either side, on account of the pains in his legs. Since August 1881 he can stand only when supported on either side. Bones and joints are normal; no spinal tenderness. Muscles very flabby, the gastrocnemii especially having wasted recently. Patient says that they have decreased. Integuments bloodless and flabby. Pigmented round scars, occasionally adherent to the periosteum, and from one to three cms. in diameter, are scattered over the shins—said to be the remnants of abscesses twenty-five years ago. Nails thick and laminated, and looking somewhat like split nutshells. Motion of arms free and precise. In the lying posture legs raised, or drawn up with effort, the leg movements being jerky, precipitate and excessive. On trying to touch the big toe of one foot with the heel of the other, patient shoots out the leg so wildly as to bring it into collision with bystanders, whilst he fails to reach the desired spot.

The cremasteric and abdominal (tactile) reflexes weak and delayed. Latent time of musculo-cutaneous "pain-reflexes" very protracted.

Right big toe, latent period	1·414″
Left ,, ,, ...	1·916″
Right index finger, latent period	0·147″
Left ,, ,, ...	0·137″

Knee jerk absent; cutaneous vaso-motor excitability slight. Sensibility to pain deficient in the legs, and trunk below level of nipples, becoming less from above downwards so that the soles are insensible even to severe pricks. Great delay in transmission of painful impressions. Tactile sensibility is not so deeply affected. The contact of a hair pencil is least felt at the soles and palms. Thermic sensibility is greatly diminished; no difference in temperatures between 8° and 20° R., nor between 20° R. and 29° R. noticed, but 33° R. and 36° R. are discriminated. The muscular sense and sense of position are entirely lost in the lower extremities. When patient's legs are lying parallel to each other he thinks they are crossed and *vice-versâ*. He cannot tell the position of his leg when it is raised to an angle of 45°. The muscles of the lower extremities react feebly to induced currents; those of the arms do so more readily. Galvanic reactions of muscles and nerves weak though normal in their order of occurrence. Besides pains there are formication and numbness in the legs and alternate feeling of cold and heat in the fingers of both hands. Bowels act only every five or six days; scybala; urine pale, alkaline, sp. gr. 1028, no sugar, but a $\frac{1}{4}$ per cent. of

albumen, and traces of indican, besides pus corpuscles and vesical epithelium. Micturition takes place from fifteen to twenty times daily.

Patient sleeps badly and his appetite is poor. The organs of hearing, smell and taste are intact. Vision began to fail in 1877; at present patient cannot distinguish a window by daylight. Optic discs atrophied. Loss of sexual power since two years.

The lungs are emphysematous. The heart is enlarged in the transverse direction; the sounds at the apex badly defined, impure; over the aortic area a pre-systolic murmur is heard. The radial and temporal arteries are thickened; the latter are also tortuous. The pulse (84) is hard, incompressible. The liver projects below the costal margin but does not seem to be hypertrophied.

Diagnosis.—Tabes dorsalis, emphysema of the lungs, dilatation of the heart, arterio-sclerosis.

In consequence of the changes in the vascular system I did not venture to suspend this patient, but resolved to try the effect of treatment by extension after Volkmann's method. The patient lay on an inclined plane with his feet raised. Cords were fastened to the feet and then passed over pulleys fixed at the foot of the bed. To the ends of the cords weights were attached. After two weeks' treatment the weights were increased to 10lbs.; the patient no longer complained of pains in the legs. This treatment was carried on in my ward from June till December 10th 1882. Pains were absent during the whole of this period. The patient was transferred to the infirmary where the treatment was continued. Slight return of the pains in March 1883. Sleep and muscular power of the legs improved, but the other symptoms remained much as before.

Case 4.—A. F., publican, at Akerman, aged forty-nine, married, seven children. No syphilis nor drink nor traumatism. No illness, with the exception of an acute attack (the nature of which is unknown) in 1854, after which his right hand was frost-bitten and lost all its fingers except the thumb.

Present illness.—In November 1881 patient drove in a snow-storm, the temperature being below the freezing point. On the road he felt hot and had abdominal pains. The fever continued after he reached home, but the pains soon left him. Twelve days afterwards he was feeling better though weak, when he had severe shooting pains in both arms and in both legs. During four days the pains increased, whilst the limbs became weaker, so that on the fifth day he could not stand nor feed himself. The

pains although less intense continued until September 1882. During that period patient had not been free from pain for a single hour. Changes in the weather had no influence on the character nor on the intensity of the pains. Motion in the legs disappeared completely at the beginning of March, 1882. According to patient's statement there was no great alteration of sensibility in the upper extremities, but he could not feel when his feet or his legs were touched. During this time sleeplessness from pain, and loss of flesh; bowels and bladder normal; but sexual feelings lost since the outset.

On admission into the Odessa Hospital on the 15th of July 1882 the patient was found to be of good conformation, though thin and weighing only 46·2 kilos. Bones and joints are normal. Flabby, palish and wrinkled about the face. Patient can be comfortable only on his back, and is unable to sit up for more than an hour, and unless propped up with pillows, the legs lie extended with toes dropped, and no trace of voluntary or reflex motion can be detected. Patient usually rests his arms on the abdomen the elbow joints being flexed; he moves them languidly but extensively, and without any co-ordination. The elbow and knee jerks are absent; plantar reflex very weak; cremasteric and abdominal intact. No musculo-cutaneous "pain-reflexes" in the legs where the bulk of the muscles is much atrophied, the extensor femoris being least affected. Gluteal fairly normal; muscles of shoulder and arm in good condition. In the forearm the flexors are more atrophied than the extensors. Left interossii deeply affected; thenar and hypothenar muscles almost non-existent. The hands have a bird's-claw appearance.

Painful pressure points along great nerve trunks, though calf muscles are tender. Palms and on the soles is of a bright red, constantly moist; no perspiration elsewhere.

The muscular sense is normal in the arms, diminished in the legs. The sensation of locality (Weber's compass) is well preserved in all the limbs. Cutaneous sensibility to pain is undisturbed in the arms; in the legs it is diminished from the knees downwards. Tactile and thermic sensibility are everywhere normal. The muscles, in which atrophy is marked, do not re-act to the induced current; the others feebly so. The galvanic current causes nothing but a slight K O C in the less atrophied muscles. No K C C, A C C, or A O C are observed.

Patient complains of tearing and shooting pains in both arms and legs, the pains coming on in paroxysms; of formication and a sensation of weight in the lower and of a constant feeling of

cold in the upper and lower extremities. Defæcation and micturition are normal. Urine shows nothing but an increase of phosphates. In other respects patient's condition is normal, though sleep is often broken owing to pain.

Treatment began on the 11th of August, 1882. After fourteen suspensions, voluntary and reflex movements appeared in the legs.

Latent period of musculo-cutaneous "pain-reflexes"—

Right great toe	0·659″
Left ,, ...	0·671″
Right thumb	0·194″
Left ,, ...	0·247″

After seventeen suspensions the toes assumed a more natural position and the patient made attempts to walk, leaning on his bedstead. He had however great difficulty in dragging his legs one after the other. From this time the pains in the limbs ceased altogether.

After twenty-four suspensions the movements of the hands were perfectly free. On the 5th of November hyperæsthesia of the skin of the legs was observed and the patient became conscious of a new feeling—warmth. Erections also re-appeared.

After the twenty-sixth suspension the greatly atrophied muscles of the forearms and legs began to respond to the faradic current and to increase in size. The body-weight was 46·7 kilos. Dynamometer: Right foot, 50lbs.; left, 40lbs.; left hand, 40lbs. A month later, considerable increase of strength (right foot, 70lbs.; left, 65lbs.; left hand, 55lbs.).

Latent period of musculo-cutaneous "pain-reflexes"—

Right great toe	0·555″
Left ,, ...	0·541″
Right thumb	0·165″
Left ,, ...	0·176″

After thirty-four suspensions the feeling of warmth in the legs became more intense and adductor rigidity occurred, lasting all through the day and sometimes even persisting at night. Suspension was given up for a time and the spasms disappeared after three weeks.

After the fifty-second suspension F. could stand on his legs without additional support or assistance, his general condition showing the following improvements: (1) Patient can walk about the ward leaning on an attendant: when in the lying posture he moves his legs about very freely; (2) he has no pains, nor (3) formication or feeling of cold in his limbs; (4) sexual

activity has fully returned; (5) arms and legs stronger; (6, 7) the muscles are larger and respond to faradisation; (8) the musculo-cutaneous "pain-reflexes" have re-appeared.

I have studied the effects of this mode of treatment in nineteen other cases, namely: lateral sclerosis (3); chronic disseminated myelitis (1); chronic meningo-myelitis (1); sciatica (1); disseminated insular sclerosis (1); tabes dorsalis (12).

The cases of tabes were the only ones in which any benefit from the treatment was observed; the others did not appear to be influenced in any way. In all the tabetics, except two, the improvement was more or less striking, and bore chiefly upon the pains and other paræsthesiæ; and on the muscular power and co-ordination of the limbs. The two cases in which no benefit from the treatment was derived were recent ones (early or "neuralgic" stage). One of rapid diminution in the use of the legs and retention of urine were observed, but even here also the pains decreased in severity.

While carrying out this treatment, I had many opportunities of observing its well-marked effects upon the characteristic lightning pains. All the patients previous to suspension suffered severely from such pains, especially during certain atmospheric changes. Rain, storms and sudden variations of barometric pressure affected some; whilst changes in the direction of the wind (N. and N.E. winds being specially harmful) had greater influence upon them. Such atmospheric perturbations were frequent at the very times of the year when these cases were under observation; and I noticed that those tabetics who were treated by suspension escaped in a considerable measure the effects of such atmospheric changes as caused considerable exacerbation among those who were not subjected to the treatment.

I now pass to the consideration of the first question, namely, what are the relations of the spinal cord, with its membranes and the nerve roots, to the vertebral canal during the process of suspension. In order to answer it on post-mortem evidence I removed the spinous processes of the fifth and sixth dorsal vertebræ, taking care to interfere as

little as possible with the ligamentous and muscular connections of the bones. The fifth and sixth nerves could then be seen passing nearly horizontally through the dura mater and forming a slight convexity directed downwards. On suspension the nerve roots became nearly vertical, forming an acute angle with the spinal cord. The body in a horizontal position measured 145cm., and when suspended 151cm. The increase in the length of the vertebral column alone from the second cervical to the fourth lumbar vertebra was 2·25cm. When the body was horizontal, skin, bone and dura mater were all marked at the same level in several places. On suspension the marks on the skin and bone were found to be 14mm. lower than those on the dura mater. Although the strain on the nerve roots was not marked it appeared to me that it was greater on the posterior than on the anterior one. The tension of the liquid surrounding the spinal cord was also increased. To satisfy myself that the opening of the vertebral canal and the removal of the two spinous processes had no influence on these results, I repeated the experiment on a body in which everything had been left intact, and found that the elongation of the whole body and of the vertebral column in particular was about the same as before.

The observations of Merkel have long since taught us that the length of the human body varies daily. According to him the increase in length after a night's rest in the horizontal position may be equal to two inches. The erect position, on the contrary, causes a shortening, as observed by Cruveilhier, Merkel and Malgaine. Suspension, as we have just seen, causes considerable increase in length. Measurements made on living subjects gave the following results in centimetres :—

NAME.	Length from superior border of spine of second cervical vertebra to superior border of spine of fourth lumbar vertebra.		Length from superior border of spine of fourth lumbar vertebra to sole of heel.		FULL HEIGHT.		DIFFERENCE.
	STANDING.	SUSPENDED.	STANDING.	SUSPENDED.	STANDING.	SUSPENDED.	
M.	47·0	48·5	89·0	90·5	151·5	154·5	+ 3
T.	51·0	52·0	99·5	102·5	164·5	168·5	+ 4
K.	60·5	62·0	93·0	96·0	168·5	73·0	+ 4·5

In taking these measurements I made use of an apparatus resembling Weber's compasses. The subject when standing was placed in such a position that the heels, sacrum, spine and occiput all touched a vertical measuring rod. A movable piece at right angles to the rod and riding upon it was then lowered till it touched the patient's head. The measurements during suspension were taken by means of two pointers, which could be moved along the measured rod and placed in contact with the desired spots on the patient's body.

The figures in the above table show that the vertebral column takes part in the general elongation. That this is not due to the straightening out of the curves alone is easily shown by taking the measurements with a tape applied to the body throughout the whole length of the vertebral column.

The extent of the lengthening depends on the extensibility of the muscles and ligaments. The greatest elongation I ever noted during suspension was 5cm., reaching 6cm. on attaching additional weight to the feet. The elongation of the vertebral column is not noticed in all subjects, in spite of an increase in length of the whole body. It may retain its ordinary length, or may even become shorter by 1 or by 1½cm, as sometimes happens at the beginning of a course of suspensions, while the patient is still unaccustomed to the method and contracts some of the neck, shoulder and trunk muscles (trapezius, latissimus dorsi, serratus magnus, rhomboids, erector spinæ). A badly fitting head-piece may also interfere with the lengthening of the spine by obliging the patient to bend his neck too much.

Before proceeding to the consideration of the *modus operandi* in suspension, I wish to say a few words concerning the *technique* of the method and also to consider its effects on the respiratory and circulatory functions.

The apparatus used is the one devised by Sayre for the application of plaster jackets, the only difference being that I do not use the second smaller cross bars. The head piece is attached to the hooks at the ends of the large bar, together

with the shoulder straps. The posterior part of the collar should be more elevated than the anterior—the patient's ears remaining free in the triangles formed by the collar and the straps.[1]

It is also necessary to remark that the treatment should always be carried out by a *medical man*, and not entrusted to inexperienced persons. The patient should be raised and lowered slowly and without jerks. Before raising it is well to see that the strain on the head and shoulder straps is equal, as in that case the patient is able to endure the operation for a longer time by transferring his weight alternately from his head to his shoulders. The first suspensions should not last longer than from one to two minutes.[2]

Suspension influences both the respiration and the circulation. The former becomes costal in type and is accelerated. The patient K., who breathed from sixteen to eighteen times per minute while standing, breathed from twenty-two to twenty-four times per minute on being suspended. In other cases a similar acceleration was noticed; on an average it was equal to four per minute. The pneumo-manometer also showed that both respiratory acts were interfered with. In the case of the same patient, while standing the pressure was equal to —30 mm. of mercury for inspiration and to + 80 mm. for expiration; while the patient was suspended

[1] Dr. Motschutkovsky has recently modified the suspension apparatus. The collar not unfrequently compresses the trachea and the great blood vessels of the neck, more especially in stout persons; this gives rise to attacks of syncope, which may assume a dangerous character, to vertigo and to cyanosis. In the new arrangement the collar is replaced by a pad and a chin piece, both connected by straps. The pad is made of hard leather and is applied to the occiput. The chin-piece is also made of hard leather, is cup-shaped, and its concavity is well padded and lined with wash leather. The chin of the patient is received into the hollow. The occipital pad and the chin-piece are connected by means of straps, which pass over the rami of the lower jaw and over the mastoid processes. In this manner compression of the blood vessels of the neck is avoided. The chin-piece can be raised or lowered by means of straps in such a way that the head remains in an easy position, which is a point of the utmost importance.—TRANSLATOR.

[2] In order to avoid attacks of vertigo and syncope during suspension, Dr. Motschutkovsky is accustomed to pay close attention to the state of the patient's consciousness. He does this by conversing with him.

It is important that the patient should not be allowed to rotate about a vertical axis: this is apt to happen when the ropes used are new.—TRANSLATOR.

the figures were —10 mm. for inspiration and + 60 for expiration.

The pulse rate is also accelerated. In the same patient K., it was equal to sixty-four beats per minute while standing. During the first minute of suspension it was 75 per minute, during the third it was 82, during the fourth 84, during the seventh 86, during the ninth 86.

Sphygmographic tracings show that the blood pressure is raised during suspension: the diastolic wave noticeably approaches the apex of the systolic wave. As the shoulder straps interfere with the circulation in the radial arteries these observations were carried out on the femorals.

From these meagre data it is, of course, impossible to come to any conclusion as to the reason why suspension does good; nor is it clear why it should prove most useful in the treatment of tabes. It is certain that during suspension changes take place in the relative positions of the spinal cord, the nerve roots and the vertebral canal, although the nerve roots do not become noticeably tense. It is probable, moreover, that, as the lower extremities become elongated to the extent of more than 3 cm., the peripheral nerves are stretched, as they are in the operation of nerve stretching. Accordingly, suspension might be supposed to act by effecting dynamic changes in the peripheral nerves after their emergence from the vertebral foramina.

This might be admitted, if the treatment were efficacious only in arresting the progress of the disease. But as some of the symptoms are markedly improved, it appears to me improbable that this can be due to changes in the nerves alone. In all slowly progressing degenerations of the central nervous system it appears to me that improvement can only be brought about by improving the nutrition of those parts of the tissue which are as yet only slightly changed; whether this takes place directly or by increasing the activity of the collateral circulation. Of late novel views as to the physiological pathology of tabes have begun to obtain ground. According to these views (Adamkiewicz) the proximate cause of degeneration of the posterior columns is a change which takes place in the blood vessels. Microsco-

pical researches have shown, that the changes in the tissue begin as foci (which afterwards become blended) situated in the neighbourhood of diseased vessels. It is obvious that, if the nutrition of the affected parts can be maintained by increasing the blood-pressure, these parts may retain their activity for a longer or shorter period. We have seen that during suspension the blood pressure is increased; the blood vessels are at the same time stretched. May it not be that these changes increase the activity of the circulation in those vessels of the cord, which are as yet unaffected?

It is remarkable that recent cases of tabes are not benefited to the same extent by suspension as those of old standing. I am of course far from claiming that tabetics can be cured by means of suspension. But on comparing the results of nerve stretching with those obtained by suspension, I must give preference to the latter. Moreover, suspension does not interfere with the patient's occupation, is not painful, and if certain precautions be observed, is not dangerous. It is true that by means of nerve stretching pain has been relieved at once in several instances. But although this is effected more slowly by means of suspension, the analgesic effect of the latter mode of treatment is most marked. None of those trophic disorders which sometimes follow nerve stretching (probably owing to irritation of the grey substance of the spinal cord) have been noticed after suspension.

The conditions which I have considered to contra-indicate suspension are as follows:—

1. Heart disease and disturbances of compensation.
2. Sclerosis and aneurysms of blood vessels.
3. Marked emphysema of the lungs.
4. Cavities in the lungs and liability to hæmoptysis.
5. Previous epileptic, apoplectic and epileptiform attacks.
6. Profound anæmia and a tendency to syncope.

Patients should not be suspended too often (not oftener than every second or third day), nor for more than ten minutes at a time.[1]

[1] As at present practised, the suspension should not, as a rule, be prolonged for more than about *two* minutes at a time.—ED.

Frequent (daily or twice a day) and prolonged (twenty minutes) suspensions gave rise to the following bad effects:—

One patient complained of sudden loss of power in the lower extremities. Some had pains in the waist and back. Others suffered from vertigo, loss of appetite and drowsiness. A small hæmorrhage into the sclerotic took place in one patient. Retention of urine was observed in one case. Finally, the patient F. suffered from spasmodic contractions of the lower extremities (which did not, however, last long).

I have not been able to decide whether the ætiology of tabes have an effect in rendering some cases more amenable to treatment by suspension than others.

In order to avoid fallacies I used no other method of treatment at the same time as suspension.

The sole purpose of this paper is to register results obtained in the treatment of one of the most painful and hitherto most hopeless of all diseases. I look forward to far more extended and varied investigations in order that the general applicability and definitive value of the method here described be brought into the light and invested with scientific certainty.[1]

NOTE.

Up to the present time the field of application of suspension has not been greatly extended in Dr. Motschutkovsky's wards, but his former conclusions have been repeatedly confirmed by more recent observations. Particularly good results have been obtained in cases of peripheral neuritis, more especially of the sciatic nerve. Among the tabetics who have lately been treated by suspension, one in particular gave evidence of remarkable improvement. This patient committed suicide later on. Dr. Motschutkovsky and myself are at present engaged in studying the histological changes of his nervous system; the results of our investigation will be published shortly. A series of observations have been made in Dr. Motschutkovsky's ward, shewing that suspension acts benefi-

[1] This article was published in 1883 in the Russian paper *Vratch*, but has till now remained inaccessible to the great majority of medical men. The translation has been revised by the author; several unimportant passages have been omitted, a few trifling errors corrected and one or two additions made by the author and myself.—TRANSLATOR.

cially in cases of impotence of non-tabetic origin. This subject will be treated by Dr. Feldman in a paper which he is at present preparing for the press.—TRANSLATOR.

AUTHOR'S NOTE, JUNE, 1889.

I have been able to obtain accurate information as to the subsequent history of these patients only in four instances:—

1. The patient, P. M., who is described as case No. 1, is at present following his occupation of assistant to a carriage proprietor. He lives at the distance of more than one verst (two-thirds of an English mile) from the place where he works, and walks there every day. During two summers he was in the habit of going on foot daily to bathe in a salt lake at the distance of about five versts from his residence. His gait is still ataxic, and he is subject at times to various paræsthesiæ, but has not suffered from pains in the extremities. Sexual activity is present. There is no patellar reflex and the pupillary reflex is also absent. There has been no relapse of any kind, although the patient has not been suspended since he left the hospital.

2. The patient, A. F., described as case No. 4, considers himself to be completely cured; he is able to work as before. He continued to improve rapidly after he left the hospital, and since that time has not been treated in any way.

3. One of the twelve tabetics who are mentioned in the table, has remained free from lightning pains in the extremities and from incontinence of urine ever since he was treated by suspension. The patellar and pupillary reflexes are both absent.

4. Finally, another tabetic, also included in the table, was under observation for two years after his course of treatment was completed. He then removed to Moscow and has not since been heard of. During that period there was no recurrence of gastric crises, from which he used to suffer at least once or twice every week. His gait also continued to be fairly good. The rapid progressive deterioration of his eyesight, which existed previous to treatment, was arrested. The patellar reflex remained absent.

With regard to the question whether a second course of suspensions might not be necessary in some instances, I may state that I have not as yet had occasion to resort to such a measure.

Clinical Cases.

CASE OF TUMOUR OF THE RIGHT TEMPORO-SPHENOIDAL LOBE BEARING ON THE LOCALISATION OF THE SENSE OF SMELL AND ON THE INTERPRETATION OF A PARTICULAR VARIETY OF EPILEPSY.[1]

BY J. HUGHLINGS JACKSON, M.D., F.R.C.P., LL.D., F.R.S., AND CHARLES E. BEEVOR, M.D., F.R.C.P.

Emily M., a widow, aged 53, was admitted under our care (National Hospital for the Epileptic and Paralysed), November 25th, 1887; she died December 31, 1887. For nearly all of the notes of the case we are indebted to Dr. Hull. There is nothing very noteworthy in her family history, nor, until this illness, in her personal history. She said that she had had a polypus removed from the uterus some years ago. The first thing in her present illness, about thirteen months before admission, was the occurrence of epileptic fits. As these attacks were very odd, independent accounts of them were obtained by Dr. Beevor, Dr. Hull and Dr. Hughlings Jackson; there was no essential variation, if indeed any, in the several accounts.

The first account given is by the patient's sister; some of the facts stated were of course obtained by her from the patient. The patient was a cook. In the paroxysm the first thing was tremor of the hands and arms; she saw a little black woman who was always very actively engaged in cooking; the spectre did not speak. The patient had a very horrible smell (so-called "subjective sensation" of smell) which she could not describe (*vide infra*). She had a feeling as if she were shut up in a box with a limited quantity of air (probably this was only her way of speaking of a feeling of suffocation; see Dr. Hull's note of the paroxysm). She would stand with her eyes fixed and directed forwards for a few moments, and then say, "What a horrible smell!" The patient did not, so her sister reported, lose consciousness, but remem-

[1] Read before the Medical Society on February 18th, 1889.

bered everything that happened during the attack; she turned of a leaden colour. The patient told us that she passed her urine in the seizures. There was no struggling, and the tongue was not bitten. She never believed the spectre to be a real person. After leaving her kitchen work she had paroxysms with the smell sensation, but no spectre. She had had these paroxysms ever since, sometimes three in a day, sometimes one in two days.

The patient herself gave to Dr. Beevor, before her admission, and to Dr. Hughlings Jackson and Dr. Hull afterwards, essentially the same account of the paroxysms with the spectre and smell. The following is re-description in essential features.

Dr. Hull writes that she said that in the paroxysms she used to see a little black woman who was rather agreeable and was always flitting about the kitchen; she always saw the same woman in every paroxysm. She never thought it was anything but a vision, but was much worried about it. When she had a fit, she used to have a very nasty smell—"burning dirty stuff"; the smell rising after the fit made her feel *suffocated*.

The patient's memory had been getting worse for six or seven months, but no other mental symptoms were noted except those which were paroxysmal—those already stated. It is to be particularly noted that she had not complained of headache during her illness—a noteworthy fact in a case of large cerebral tumour; she had pains in the neck for the last week or two. Her illness had been preceded by trouble, loss of money—not a likely thing to have been the cause, or, at any rate, more than the exciting cause, of any such symptoms as she had. A week before admission she was noticed to get gradually weak of the left side, and it was observed that the face was "lop-sided," and the left eye was noticed to close. She complained of a leathery feeling on the left side of the body.

On admission, November 25th.—A very fat woman; she lay mostly in a torpid condition. After a good deal of rousing she could be made to walk and could walk fairly well when urged along, yet without decided paralysis of the limbs there was a tendency to go to the left. The patellar, triceps and wrist jerks were equal on the two sides; there was no ankle clonus; the right plantar reflex was greater than the left, but both were active. There was a little dropping of the left side of the face, especially seen when she spoke. The tongue was protruded slightly to the left; it was not tremulous. There was no ocular paralysis, no nystagmus; the pupils were of equal size and their reactions were normal. There was no hemianopia. Sensation,

tested by light touches and by pin pricks, appeared to be normal and equal on the two sides of the body. The optic discs were normal. A few days later (December 1st), it was found that she could read fairly well with spectacles, but did so as if it were a great trouble, and spoke in a slipshod, slovenly kind of way. (There was never any aphasia, nor any real difficulty in articulation; nothing more in her speech than inertness, as in all else.) Taste and smell were normal. To return to the account of November 25th: there was no obvious defect of hearing, she being able to hear on both sides. No trustworthy comparison between the ears could be made on account of her condition. There was no visceral disease discovered, and the necropsy revealed none. It may be well to say that she kept her bed until death, except that she was occasionally got up, as will be mentioned, to test her legs; as will be seen, she had the delusion that she got up to walk at other times.

November 27th.—She disturbed the other patients in the night for some time by calling out for chops. In the morning she did not remember anything about it.

December 1st.—It was to-day that her reading was tested and that hemianopia was excluded. She walked half the length of the ward, but had to be supported; she leant heavily against the table and chairs, and would, unhelped, have fallen; she made no attempt to save herself. She took food well: she said she had no pain anywhere.

December 8th.—She said that this morning she had a fit, and that it consisted in a suffocating feeling; she had also a "nasty dreadful smell," but she could not describe it more.

December 9th.—This morning, at 7 a.m., she said she wanted to get up and go out. She said she was convinced that she was perfectly able to walk, and said that she did walk yesterday. She said that she sometimes got up, and she complained that she was flung back on her bed; she spoke in an aggrieved way. When got up it was found that she did walk a little better than when last tested; she still has a marked tendency to fall to the left, and would fall to that side if not supported. She says, notwithstanding, that she is quite able to walk.

December 19th.—She says that she thinks she went for a walk yesterday. Is very lethargic this morning and can scarcely be got to say anything. No optic neuritis. Urine acid, no albumen.

December 23rd.—The patient has been getting into a still more torpid condition the last few days. The left arm and leg

are almost perfectly powerless; passive movement of the leg causes pain. Sensation not absent, but difficult to estimate on account of the patient's condition. No optic neuritis.

December 27th.—Is still very drowsy. Her left side seems to be quite paralysed, but no loss of sensation is made out. To-day Mr. Gunn examined the patient's eyes. The examination was difficult on account of the patient's condition, and from mucus on the cornea. She was asleep when the examination was begun, and her pupils were contracted to a small (normal) size; shortly afterwards she became partially aroused, and then the pupil suddenly dilated to the same size that it had been some hours previously under atropine. It did not contract to light. There was well-marked double optic neuritis; the edge of the right disc was undefined; the outer edge of the left was still determinable; there was not much swelling, and there were no hæmorrhages.

December 31st.—After only a gradual worsening, she died to-day at 3 A.M.

Necropsy, January 2nd, 1888 (thirty-five hours after death). There was a small sub-peritoneal fibroid, about the size of a hen's egg, of the uterus. Beyond this nothing noteworthy was found except in the head. The brain and cord were given to Dr. Beevor for investigation.

EXAMINATION OF THE BRAIN.

BY DR. BEEVOR.

THE patient, a description of whose case Dr. Hughlings Jackson has just read, first came under my care as an out-patient at the Queen Square Hospital. She there described to me the aura which Dr. Jackson has detailed, the nasty smell followed by a sense of suffocation, and the vision of the little black woman who was very busy about the kitchen. The combination of these two things sounds so fantastic and almost absurd, that I feel pretty sure that one would be very liable to overlook their importance, or to consider them to be merely the utterances of an hysterical patient, if Dr. Jackson had not impressed upon us in his writings the absolute necessity of accurately recording all the details which a patient gives of the warning preceding an epileptic seizure, however trivial they may seem.

It is interesting to note with regard to the co-exist-

ence of the visual aura and the sense of suffocation with this sense of a horrid smell, that Dr. Gowers, in his work on "Epilepsy," p. 62, states, "that the only associations noted (with the olfactory aura) were, with a visual aura (two cases) and with a feeling of suffocation (two cases)."

After the patient had been an out-patient for a few weeks she became worse, and was admitted under Dr. Jackson.

As the case has been so fully described from its clinical aspect by Dr. Jackson, I need not further allude to it.

On examination of the brain, it was seen that the right temporo-sphenoidal lobe was the seat at its most anterior extremity of a tumour of the size of a tangerine orange. The growth was seen to involve the extreme tip of the temporo-sphenoidal lobe, and especially the part of it which is in front of the uncus of the hippocampal or uncinate convolution, and which contains the structure known as the nucleus amygdalæ.

This extreme anterior end of the temporo-sphenoidal lobe is called the pyriform or hippocampal lobule, and has been found by Broca to be very much developed in animals with a keen sense of smell, such as cats, dogs and rabbits, and to be quite rudimentary in animals like the dolphin, which have very little powers of smelling.

According to Dr. Ferrier's experiments ("Functions of the Brain," p. 320), he states that the "affections of smell and taste are evidently related to lesions of the hippocampal lobule and the neighbouring regions."

The exact position of the growth will be more clearly shown by describing the appearances seen when frontal (*i.e.*, transverse vertical) sections are made across the brain.

On making a frontal (transverse vertical) section through the brain at the level of the optic chiasma (Fig. 4) the whole of the anterior end of the temporo-sphenoidal lobe of the right side was found to be occupied by a tumour; it involved the nucleus amygdalæ and the central white matter of the temporo-sphenoidal lobe; but it did not affect the grey cortex of the uncinate convolution (hippocampal convolution), or of the first temporo-sphenoidal convolution. The

nucleus lenticularis was much compressed and flattened by the growth, and the internal capsule seemed to share in the compression. On making a frontal section of the brain at the level just in front of the pons (Fig. 5), all the central white matter of the right temporo-sphenoidal lobe was seen to be involved. The lesion lay just outside the descending cornu of the right lateral ventricle. The cortex and white matter of the hippocampal and first and second temporo-sphenoidal convolutions were not affected; the cortex of the third temporo-sphenoidal convolution was not involved, but its central white matter was partly invaded by the growth.

At the level of a frontal section through the middle of the pons (Fig. 6), the lesion is seen about the size of a threepenny piece just outside and below the descending cornu of the lateral ventricle and the cornu ammonis; it involves the inferior part of the central white matter of the temporo-sphenoidal lobe, but it does not extend to the convolutions or their central white matter.

Behind this point the lesion rapidly diminished in size and was hardly visible in the next section, which was made about half an inch behind the preceding one.

On microscopic examination by Dr. Colman, the resident medical officer to the hospital, the growth was found to be a small round-celled sarcoma.

It will thus be seen that we have here a tumour which is localised in one spot, namely, the extreme anterior end of the right temporo-sphenoidal lobe, the part which is called the hippocampal lobule and which contains the nucleus amygdalæ. This region is largely developed in animals with a keen sense of smell, and in his experiments Dr. Ferrier has found in monkeys that this part of the brain is associated with the sense of smell. I think we are therefore justified in thinking that the sensation which the patient complained of, viz., "the horrid smell," was produced by the irritation of the grey matter of the olfactory centre in the right hippocampal lobule, and it is interesting to note that this centre was probably not entirely destroyed, as is shown by the fact that the sense of smell was not abolished on that side, and that had the whole olfactory centre

been destroyed, there would not have been any cells remaining to register the irritation caused by the growth, and to produce in the mind of the patient the sensation of a "horrid smell."

The growth had also pressed upwards upon the right lenticular nucleus and the internal capsule of the same side, and this would account for the weakness of the left side of the tongue and the protrusion of this organ to the left, and also for the drooping of the left side of the face—symptoms which were noticed on the patient's admission. It is important to note that although on admission there was no absolute paralysis of the left arm and leg, they subsequently became completely paralysed, due, no doubt, to the gradual growth of the tumour. The fibres in the internal capsule which pass down from the cortex for the face and tongue are more anterior in the horizontal line than those for the limbs, and this would perhaps account for the face being affected before the limbs.

Remarks by Dr. Hughlings Jackson.—This case interests me especially as presenting epileptic fits with what I call the "dreamy state" (commonly called "intellectual aura"), in association with a crude sensation of smell. It is one of a group of epilepsies on which I remarked in a paper in 'BRAIN,' July, 1888 ("On a Particular Variety of Epilepsy: Intellectual Aura"). The case is referred to in a foot-note on the first page of that article.

The case is of great value as evidence bearing on localisation in the cortex of the anatomical basis of the sense of smell; it confirms Ferrier's researches. I leave this aspect of the case for my colleague, Dr. Beevor, to comment on. I would, however, remark, that had there been no crude sensation of smell (physically no paroxysmal discharge of olfactory nerve elements) the case might have been thought of as discountenancing Ferrier's localisation. But the case shews that not only the effects of destructive lesions, but those of discharging lesions also have to be considered with regard to sensory localisation, just as both are considered with regard to motor localisation.

I refer to my paper in 'BRAIN,' July, 1888, for further

remarks on the variety of epilepsy, of which this case is one example. I once more draw attention to Dr. James Anderson's report of the case of one of his patients, who had this variety of epilepsy and a crude sensation of taste—'BRAIN,' October, 1888.

Remarks by Dr. Beevor.—Besides the case reported by Dr. James Anderson, to which reference has already been made, Dr. Ferrier has referred us to two cases in which there were olfactory symptoms during life due to intracranial growths. The first case is one by Sander,[1] described as "Epileptische Anfälle mit subjectiven Geruchs Empfindungen bei Zerstorung des linken Tractus olfactorius durch einen Tumor." Here the patient had fits which were preceded by the warning of a "dreadful disagreeable smell," and he then had chewing movements of the jaws and spitting of saliva,[2] and later on he had convulsions about the face but not in the limbs. The patient became blind, and his mental condition very obtuse, so that it was never possible to test his sense of smell on the two sides. On post-mortem examination there was found, in the middle fossa of the skull, a tumour (glioma) the size of half a large apple, situated on the under surface of the brain, at the border of the left frontal and temporal convolutions. Half the growth involved the anterior part of the temporo-sphenoidal lobe, the other part reached anteriorly across the fissure of Sylvius, so as to involve completely two gyri of the frontal lobe (which two gyri is not stated); inwards the growth reached the middle line. The important point was that the left tractus olfactorius was intact only at its anterior part, but posteriorly it was involved in the growth with the left optic nerve and tract. Part of the growth affected the base of the left temporal lobe and grew into the brain substance below the lenticular nucleus, and another part on the inner surface of the lateral ventricle involved the left ammon's horn.

In the above case, although the anterior end of the

[1] Archiv. f. Psych., 1874, vol. iv., p. 234.

[2] Dr. Hughlings Jackson has frequently drawn attention to the occurrence of the "dreamy state" with chewing movements and with spitting. In Lander's patient, and in the other cases referred to in my remarks in the text there is no mention of a "dreamy state" (intellectual aura).

temporo-sphenoidal lobe seemed to have been involved, the growth was so large as to have destroyed the left olfactory tract, so that it would be impossible to say whether the attacks were not caused by pressure of the growth on this tract; it was also impossible to ascertain whether the sense of smell was intact on the two sides.

Sander refers to other cases by Lockemann,[1] Westphal,[2] Schlager,[3] in which olfactory auræ preceded the fits, but in all these cases the olfactory bulb or tract was affected either by tumours growing either in them or from the overlying frontal lobes.

In none of these cases did the lesion occur in the temporo-sphenoidal lobe without involving the olfactory nerves, and they do not afford any direct evidence of the localisation of the olfactory centre in the tip of the temporo-sphenoidal lobe.

The second case referred to by Dr. Ferrier is one by Dr. McLane Hamilton—"On Cortical Sensory Discharging Lesions (sensory epilepsy)."[4] Here a woman, aged forty, had attacks dating from her tenth year, when she had a fall on to her head. In the attacks she frothed at the mouth, became livid and was convulsed for a great time, but before the fits she nearly always had a peculiar aura: "She suddenly perceived a disagreeable odour, sometimes of smoke, sometimes of a fœtid character, and quite uncomplicated by other sensory warnings. She compared it to the smell of burning rags or the smell of a match, and 'it sometimes rose up her head and choked her.'" Two years later she died from phthisis, and in the brain a low-grade form of hæmorrhagic pachy-meningitis was found about the base of the brain. The most marked changes were found in the lower part of the right temporo-sphenoidal lobe, where a decided shrinkage of tissue was seen, with depression and adhesion of the pia mater, the induration involving the uncinate gyrus and parts of the adjacent convolutions. The olfactory nerves were not involved, nor was the third frontal convolution. From the sketch of the brain

[1] Zeitschr. f. ration Med., Vol. XII., p. 340.
[2] Allg. Zeitschr. f. Psych. Vol. XX, p. 485.
[3] Zeitschr. der Gesellsch. d. Aerzte zu Wien, 1858, Nos. 19-20.
[4] *New York Medical Journal*, 1882, Vol. XXXV., p. 575.

given in this paper, the lesion seems to have involved the inferior surface of the temporo-sphenoidal convolution extending to the anterior end, and affecting especially the third temporo-sphenoidal and the uncinate convolutions; but to what extent this latter was involved is not mentioned, nor is it stated whether the disease involved the uncus itself, the cornu ammonis, the hippocampal lobule, or the inner surface of the temporo-sphenoidal lobe.

The last case is exceedingly good for the purposes of localisation, as the lesion, as far as it is described, was very definite, and the olfactory nerves were found to be intact. Unfortunately, however, there is no mention as to the condition of the sense of smell on the two sides during life.

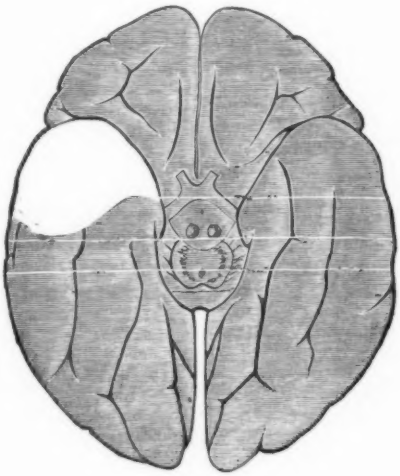

FIG. 1.

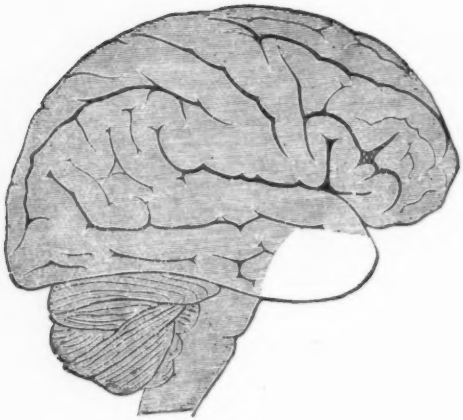

FIG. 2.

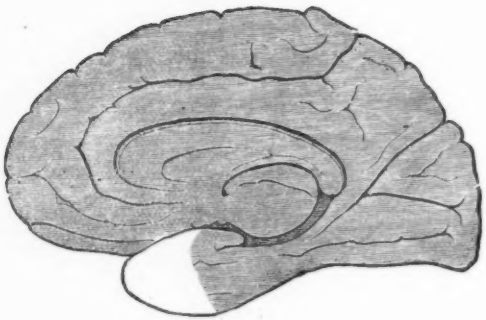

FIG. 3.

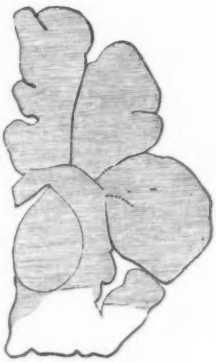

FIG. 4.

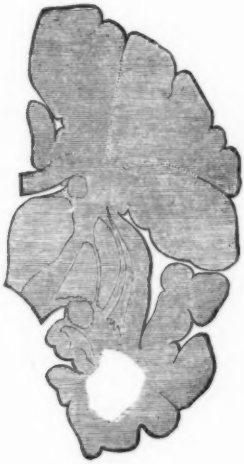

FIG. 5.

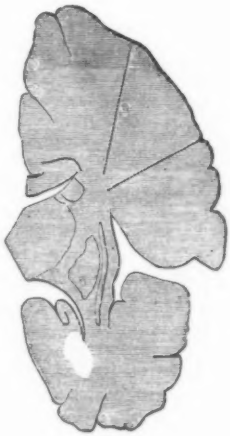

FIG. 6.

EXPLANATION OF THE FIGURES.

The part occupied by the tumour is shown as it appeared on the surface of the brain, and also in frontal sections.

The horizontal transverse lines ruled across Fig. 1 indicates the levels at which the frontal sections, shewn by Figs. 4, 5 and 6, were made.

Critical Digests.

CEREBRAL LOCALISATION IN ITS PRACTICAL RELATIONS.

BY CHARLES K. MILLS, M.D.

(*Continued from p.* 288.)

A CAREFUL, elaborate, clinical study of hemiplegias, monoplegias and aphasias will eventually enable us to separate with considerable certainty lesions of the cortex from those of the centrum ovale, capsules, and ganglia. We will do this by relating the symptoms found not only to lesions of the cortex and the projection system of fibres, as is too commonly the restriction placed on our studies in this direction, but also to lesions of commisural and association fibres. I am convinced that a lack of consideration of these commisural and association fibres is at the bottom of much of our confusion in analysing certain cases. Very few lesions are absolutely cortical. Many of those which are generally regarded as cortical involve to a greater or less extent the sub-cortex. As every convolution of the cerebral surface is connected with some other, and probably with many other convolutions, some association fibres must nearly always be destroyed in these cases.

Differential Diagnosis, particularly of Jacksonian Epilepsy—Dural and other Reflex Epilepsies.

Sufficient diagnostic difficulties are still present to make it important in the light of the tremendous impetus towards operations to carefully examine all questions of differential diagnosis. We should know in connection with cerebral motor localisation, whether certain affections do not simulate cortical epilepsy so closely as to sometimes endanger exact diagnosis. In certain motor and especially spasmodic affections for example we have striking resemblance between affections clearly of reflex origin and those as demonstrably central. Trigeminal epilepsies, whether dural, facial, dental, nasal, pharyngeal, laryngeal, or

of whatever local origin, may cause unilateral convulsions or even monospasm. Brown-Sequard has contributed largely to our knowledge of this subject both in his early and recent researches. During a few years stimulated by practical specialism, much work in the direction of diagnosis and treatment of reflex epilepsy has been done, some of the best of it by members of the Associations represented in this Congress. The same conclusion might be arrived at for all, as that of Boucheron [1] with reference to aural epilepsy, namely, that spasm may proceed from lesions of the ear, eye, nose, pharynx, larynx, face, scalp, or dura mater, and may present all the clinical varieties of epilepsy, or even a form of hystero-epilepsy; and that the point of origin of these disorders is intense excitation of a sensory nerve.

Dural epilepsies are especially worthy of attention. During a recent operation in one of the Philadelphia hospitals a faradic current accidentally applied to the dura mater almost instantly produced spasm which invaded the whole body. Dupuy [2] has published various papers regarding irritation of the dura mater causing muscular movements, claiming that his results are constant when the animal experimented upon is not in a state of anæsthesia incompatible with the manifestations of animal life, and when it has not lost too much blood. Brown-Sequard and Burdon Sanderson have recorded similar phenomena; and the facts of these experiments have been used as arguments against cortical localisations. The Committee of the New York Society of Neurology and Electrology, in 1874 found that galvanisation of the dura or other sensitive parts produced by reflex action muscular twitchings, oftenest on the same side of the body. Duret [3] has given particular attention to the rôle played by the dura mater in the production of sensory, spasmodic, and other phenomena. He does not however, with Dupuy, hold that the fact of the production of spasms in this way in the least invalidates the doctrine of cortical motor localisation, but that a clear differentiation between dural and cortical spasm can be made. Rochefontaine has shown that irritation of the dura mater determined cries of pain and general movements more or less energetic; and also that mechanical irritation of the sensitive points of the membrane produced in certain conditions movements limited to

[1] *Compt. rend. Acad. de Sc. Paris*, 1887, cv., 944-947.

[2] *Examen de quelque points de la physiologie du cerveau.* (Thèse inaugurale, Paris, 1873.) Also: *Experiences sur les functions motrices du cerveau*, 1888. *Compt. rend. des séances de l'Acad. des Sci.*

[3] '*Sur les Traumatismes Cérébraux*' and *Brain*, April, 1878.

one or several parts of the body, the movements of the limbs on the same side being more energetic than those on the opposite side. Franck has made careful comparison and contrast of cortical epilepsies and those which are reflex and toxic, including those which are due to irritative lesions of the dura mater. In one of his experiments [1] he produced an epileptic seizure from mechanical irritation of the dura, and among other things noted was that at the moment of the irritation of the dura mater the muscles of the face of the same side were attacked with violent convulsions. Attacks followed, as many as nine in twenty-five minutes, all clonic and generalised.

The following is an abstract of the record of this experiment:

"Experiment No. 45 (Jan. 7, 1879) with M. Senna of Coimbæ. Reflex attack of epilepsy (excitation of the dura mater) commencing on the side irritated.—State of disease.—Arrest of the salivary flow in the attacks.—New series of reflex attacks by incision in the skin.—Circulatory modifications in this form of epilepsy.

"A young dog, spaniel of large size, very vigorous. The motor zone was exposed at the right side while under the influence of a slight anæsthetic of chloroform; a large opening was made in the frontal sinus in order to discover the excitable region, the crucial edge forward.

"It was proposed to study at the same time with salivation, the modifications of the heart and of compression in their connection with the cortical origin of convulsions; but the animal was taken with a reflex attack of epilepsy under the following conditions: The dura mater had been cut all round the trephining point; a fragment remained adherent to the anterior inferior angle of the wound and caused a slight flow of blood somewhat interfering with the experiments at excitation. At the time when an attempt was made to stop the flow of blood with a piece of medicated cotton, the simple friction of the strip of dura mater provoked a series of violent convulsive attacks, having their point of commencement in the muscles of the face and neck on the *same side* (contrary to the epileptic attacks of cortical origin, which *always* commence in the opposite side of the body, and severely in the corresponding muscles to the cortical centre excited).

"The first attack was exclusively clonic, very violent and generalised.

"During several minutes spontaneous attacks succeeded, separated from one another by a few seconds only; at the fourth attack the convulsive movements and the salivary escape were simultaneous. It was remarked in the first periods of the attack the salivation came on slowly; it appeared in this fourth attack only thirty-two seconds after the commencement of the clonic movements.

[1] *Op. Cit.*, p. 470.

"The animal had nine grand attacks in succession in twenty-five minutes. Then exhaustion came on and it was quiet, the respiration rapid, the heart beats quick, and the arterial pressure much diminished.

"It was left to repose for half an hour, then wishing to apply a manometer to the femoral artery, an incision was made in the skin of the thigh. At this moment a new convulsive explosion came on and without pauses, in the same register as had been made in the former attacks." * * * *

The nerves of the dura mater spring from the fifth pair, and are distributed nearer to the internal than to the external surface of the membrane, which explains why some lesions of the dura are more likely than others to lead to spasm. The difference depends, in part at least on the site and intensity of the lesion with reference to the internal and external aspects of the membrane. Sub-dural hæmorrhage is more likely to give rise to reflex spasms than extravasation between the membranes and the skull, unless the blood tears through the membrane. A spicule of bone, in like manner, driven into the dura is more likely to cause reflex dural spasms than a depressed fragment; while a tumour arising in the membrane is more likely to bring about the same result than an exostosis, or a neoplasm growing from the agglutinated membranes into the brain substance, as is so often seen in intra-cranial growths.

I have notes of five cases in which operations have been performed for epilepsies apparently reflex in character. In two of these spicules of bone were removed from the dura mater. These cases bear out to some extent the views of the existence of distinctive characteristics for reflex epilepsies, but also point to certain resemblances to cases of cortical epilepsy. In one case in which fracture was present in the left frontal region, anterior to the motor area, the patient had convulsions at irregular intervals of weeks or months, usually having sharp pain at the seat of the scar before the seizure. He had no loss of sensation nor paralysis. His convulsions were frequently unilateral; I saw him in one which was confined entirely to the left side, and began in the left leg. This case was trephined for me by Dr. W. J. Hearn, of Philadelphia, and a spicule of bone dissected from the dura mater. In another case the patient was trephined by Dr. J. W. White at my request, for a fracture from a pistol-shot wound just above the right temple. Nearly three months after the injury he began to have spasms; and had had about seven seizures in all. In a convulsion which I witnessed he was completely unconscious; his body was twisted somewhat to the right; his face and all his

limbs as well as his head and trunk taking part in the spasm which was tetanic in character. In another case in which an operation was performed for me by Dr. Hearn, the spasm seemed to show a somewhat confusing admixture of what might be termed dural and cortical characteristics. Notes on this case were furnished to Dr. J. B. Roberts, and were published by him in his pamphlet on the 'Operative Surgery of the Human Brain.' In a fourth case seen with Dr. L. W. Steinbach, the patient had been subject to convulsions which seemed to date back to an injury to the head; he had a scar and apparently a depression of the skull over the frontal region. Pressure on this scar brought on a unilateral, largely tetanic convulsion on the same side as the scar. A flap including the scar was lifted, and trephining was performed, but nothing abnormal was found in the inner table or in the dura. The scar was excised. In a fifth case, the patient had convulsions, sometimes on one side, sometimes on both, and these could be brought on by pressure on a scar left by an old sabre cut. The cicatrix was cut out and the patient recovered; at least, he remained for several months in the hospital without attacks, although before the operation he had been having them at frequent intervals.

With Franck I fear we are not always able to make a trenchant separation between cortical and reflex epilepsies; but a few points may be indicated. In reflex epilepsy the attack does not begin with brusque tetanisation as in the case of cortical disease. If the reflex epilepsy has a tonic period it rises slowly to its maximum. In cortical epilepsy the convulsion begins without exception on the side of the body opposite to the side of the brain excited or irritated; in reflex epilepsy, or at least in dural and perhaps other forms of trigeminal spasm, it frequently begins on the same side as the focus of irritation. Unfortunately we have not here a radical difference, as it may begin on either side in reflex cases. In the reflex cases, if the spasm begins locally or unilaterally, there is not likely to be a definite initial or signal symptom and serial order of movement; one half of the body usually plunges immediately into spasm.

True Jacksonian epilepsies are, I believe, sometimes reflex in origin; that is, they become established as the result of intense persistent peripheral irritation, dural, dental, palmar, &c.; and even after the source of irritation is removed the cortical discharges continue. Herein perhaps lies the explanation of Jacksonian spasm in which gross lesion is not discovered,

and herein also sometimes is to be found justification for operation for the removal of cortical discharging areas, even when such lesion is not present. Such a method of origination of cortical epilepsy is in accordance with physiological principles. Meynert,[1] in the development of his idea of a projection system, has perhaps more clearly than anyone else made apparent the method in which this result may be brought about. Movements which were originally reflex in character may after a time result from cortical impulses. In the normal brain no reflex actions can be performed without exciting to action secondary volitional movements which no longer requires the stimulating influence of a reflex action. Some of the observations and experiments in hypnotism, as those of Heidenhain[2] in particular, also throw some light upon the manner in which reflex epilepsies may develop into true organic cortical disease. The phenomena of unilateral hypnosis are particularly interesting in this connection. When certain definite cutaneous surfaces are irritated, certain muscles and groups of muscles related to these areas can be brought into isolated or successive action; stroking the ball of the thumb, for example, causes adduction of the thumb towards the palm; or stimulating the skin over the sterno-mastoid causes the head to assume the wry-neck position.

A case reported by me in 1880,[3] is interesting in connection with this question. It was one of epilepsy clearly Jacksonian in type, and as clearly due to a fibroma involving a nerve trunk on the palmar surface of the hand. The patient, fifteen years old, had had the seizures since the age of four years, they coming on after an injury to the hand at the situation of the fibroma. After removal of the growth, she had spasmodic attacks of the same type as before the operation, but less in frequency for a year, after which she rapidly improved, and I have been recently informed has had no spasms for nearly two years.

A description of the usual character of the attacks shows that they were distinctly Jacksonian. The description will be quoted at length because of the importance of the matter under discussion.

The distal phalanx of the ring finger of the right hand was first flexed; secondly, a few spasmodic movements of flexion would occur in this finger; thirdly, the other fingers and thumb of this hand would begin to twitch convulsively—the second phalanges would be flexed, the last extended; fourthly, the clonic

[1] 'Psychiatry, a Clinical Treatise on Diseases of the Fore-Brain.' Translated by B. Sachs, M.D.

[2] 'Hypnotism or Animal Magnetism.' Translated by L. C. Woolridge, M.D.

[3] *Phila. Med. Times*, December 18, 1880.

convulsive movement would extend to the right hand, forearm and arm, and simultaneously the muscles of the lower part of the right side of the face would become affected with spasm, a tremor also appearing in the tongue during this period; fifthly, the right arm and leg would now become affected with a clonic spasm, causing them to assume positions of flexion, the head, neck and body being drawn by the spasm at the same time to the right, a condition of pleurosthotonus being in fact produced. The seizures would pass off with a very severe jerking movement of the right shoulder, and a renewal of the twitchings of the muscles of the right angle of the mouth. These movements of the shoulder and mouth would sometimes occur only once, just before the close of the attack; more frequently, however, they would take place two or three times in succession. Occasionally the patient would bite her tongue during the paroxysms. She apparently was never entirely unconscious during the attack, no matter how severe it might be. During the height of the convulsion, if her hand was pressed too hard she would manage to gasp out, "Don't," or to make some other exclamation.

Unilateral Nervous Affections in Bright's Disease.

Considerable evidence has accumulated to show that affections of the nervous system, strictly limited to one half of the body, occur during the course of some forms of Bright's disease. In this country Dercum[1] has reported cases of hemichorea, hemiplegia and unilateral convulsions. Raymond,[2] Chantmesse and Tenneson[3] reported series of cases of unilateral affections, chiefly hemiplegia and epilepsy apparently of uræmic, or at least renal origin. In not one, according to the reporters, could the trace of a strictly focal lesion be discovered. Chaufford[4] reports a highly-interesting case under the title of uræmic convulsions of the Jacksonian form.

Hystero-Epilepsy and Jacksonian Epilepsy.

Some cases which seem to be clearly forms of hystero-epilepsy closely resemble organic epilepsy of the Jacksonian type. Hystero-epileptic attacks, it is well known, can be produced by irritation of the hystero-epileptogenic zones, described by Charcot, Richer, and others, which are evidently analogous to the epileptogenic zones of Brown-Sequard.[5] Almost every form of spasm in

[1] *Jour. Nervous and Ment. Dis.*, vol. xiv., No. 8, August, 1887, p. 473.
[2] '*Thèse pour le Doctorat en Médecine*,' 1878, Versailles, and *Rev. de Méd.*, Sept., 1885.
[3] *Rev. de Méd.*, Nov., 1885.
[4] *Arch. Gen. de Méd.*, Paris, 1887, ii., 5-19.
[5] *Lancet*, Lond., 1886, ii., 1211-1213, Abstract of Brown's Lectures.

localisation and extent can be found in the descriptions of hystero-epilepsy. Features of distinction are however present. Undoubtedly one reason for the similarity between spasmodic affections reflex, hysterical, toxic and cerebral, lies in the fact that in these cases, whatever may be the starting point, central areas are discharged and give definite character to the convulsions. Horsley speaks of hystero-epilepsy as a cortical disease, but this view cannot be upheld for all cases, if he means by this that the spasms are usually the result of cortical discharge. They are rather sometimes bulbar or spinal, cortical inhibition being removed.

The difficulties of making a diagnosis between grave hysteria or hystero-epilepsy, and cerebral tumour or other organic lesion with apparent or real Jacksonian symptoms, is sometimes great, and was strikingly shown in a case seen by me in consultation with Dr. J. M. Barton, of Philadelphia. This patient, a married woman, thirty-five years old, came under observation in the spring of 1886. She had been in bed eight weeks, and had taken no part in her household affairs for several months. Her sickness began with complaints of headaches and feelings of slight numbness in the left hand and arm; she would occasionally drop things from this hand. She soon developed analgesia and anæsthesia in the left arm and leg, and sometimes in the face; this varied in severity. Her mental condition gradually changed, she became irritable, absent-minded, and lacking in attention and judgment. She had at times hallucinations of sight, which usually occurred after lying down, these often taking the form of animals, as cats, mice, &c., disappearing round corners. Hearing was good in the left ear, but in the right was diminished; careful examination showed no external or middle ear disease.

She suffered almost continuously from headache, which she described as violent and agonising, and which she localised mostly in the fronto-parietal region; and a cranial area over the right motor zone was very tender to pressure and percussion. Nausea and severe vomiting came on late, and both headache and vomiting were accompanied by vertiginous sensations. A curious symptom was a constant diarrhœa, the patient having from six to fifteen passages a day, and sometimes as many as twenty-four. She also often spat a bloody fluid from the mouth. Her appetite and sleep were much impaired, and she lost considerably in weight.

Ophthalmoscopic examinations made in March, 1886, by Dr. W. W. McClure, gave the following results: *Right eye*:—The pupil more active than the left; media clear; slight physiological

cupping and venous pulsation; no arterial movement observable. Slight choroidal change in pigmentation to the temporal side of the nerve; outline of nerve above not defined, but merged with the retina. Macula good. Hypermetropia. *Left eye*:—Pupil more dilated and less active than in the right eye. Appearances much like those seen in the other eye, but worse. Evidences in colour and margin of slow chronic inflammation. Macula good. Right eye, vision, $\frac{20}{50}$; left eye, vision, $\frac{20}{70}$.

After her illness had continued several months, the patient began to experience at times attacks of flexure of the fingers and thumb of the left hand, and cramp-like feelings in the forearm and arm. Later these spastic attacks began in the left foot, causing the great toe to be flexed upward and the other toes downward, with also some cramp feelings in the left leg.

A number of consultations were held with reference to this patient with Drs. J. B. Roberts and C. B. Nancrede, who took the view that the case was probably hysterical, and treatment was eventually suspended.

The patient's general health improved; she gained in weight and attended to household and business affairs, never however getting entirely rid of her main symptoms, and recently some of these have returned with renewed vigour. She has fallen several times in public places and at her own home, appearing to become entirely unconscious. The attacks are preceded by a thrill passing up the left leg, and twitching of the left arm; the latter continuing through the entire seizure and being the only muscular movement noticed.

Visual Localisation.

Next to determinations of the motor zone visual localisations are the most conclusive, and this in spite of the hard-fought battles of the physiologists over the cortical sight areas. Clinical medicine and pathology have here come bravely forward to clear away the storm. A few well-reported cases of hemianopsia with autopsies, as those by Jastrowitz,[1] Haab,[2] Huguenin,[3] Monakow,[4] Seguin,[5] Hun,[6] Féré, Keen and Thomson, seem to settle beyond doubt the connection of the cuneus and adjacent region with the retina and simple visual sensation.

[1] *Centralb. für prakt. Augenheilk*, vol. i., December, 1879, p. 254.
[2] *Klinische Monatsblatter f. Augenheilk*, xx., 141, 1882.
[3] *Ibid.*
[4] *Archiv. f. Psychiat, u. Nervenkrankeiten*, vol. xvi., S. 166.
[5] *Jour. of Nerv. and Ment. Dis.*, vol. xiii., No. 1, January, 1886, 1-38.
[6] *Am. Jour. Med. Sci.*, vol. xciii., January, 1887, 140-168.

Jastrowitz has recorded a case of paresis of the right leg, arm and face, with a peculiar form of aphasia. The patient was unable to read and write connectedly; he could not understand written words. The history does not relate definitely whether or not he understood spoken words. Right hemianopsia was also present. The autopsy showed tumour of the left occipital lobe and precuneus.

Seguin, in a contribution to the pathology of hemianopsia of central origin, in many respects the most valuable publication on the subject which has yet appeared, collected forty cases with autopsies, and five traumatic cases without autopsies. Eleven of these were cases of hemianopsia due to lesions of the white substance of the occipital lobe. Sixteen were cases of cortical lesion, or of lesion limited to the cortex and the white substance immediately subjacent; and four of the sixteen (those of Haab, Huguenin, Féré, and Seguin referred to above) are what might be termed conclusive cases as to the question of the location of at least a portion of the cortical visual centre in man; as in them the lesion was circumscribed and occupied nearly the same place in the occipital lobe. They would, at least, seem to settle definitely that in the cuneus and its immediate neighbourhood the visual half centre for retinal sensations is located. Owing to their importance I will briefly give from Seguin a condensed abstract of these cases.

Haab's case was a man, sixty-eight years old, who had an attack of temporary paresis of the left extremities. The patient complained that he could not see to his left with his left eye—though his right eye was normal. No anæsthesia; intelligence was normal, hearing good. Central vision—1 (H.2). There was left homonymous hemianopsia, the limit reaching quite (?) up to fixation point. In the right fields colour perception was good. Optic nerves presented a "senile greyish colour." The autopsy showed the extremity of the right hemisphere five mm. shorter than its fellow and a depression in the right occipital lobe, the pia hanging loosely over a cavity containing clear fluid. The patch was mostly upon the mesial aspect of the hemisphere (including apex). It occupied the site of the fissure hippocampi, and extended beyond it above and below. The white substance was but slightly injured.

Huguenin recorded the case of a girl, aged eight years, whose chief symptoms were headache in paroxysms; later, frequent vomiting, sleep broken; severe convulsions which frequently recurred; increasing dementia; slight neuritis with some swelling.

It was noticed after some months that the patient held her head obliquely to the left. Examination revealed left homonymous hemianopsia, the only symptom indicating a focal lesion of the brain. The patient died of broncho-pneumonia. At the autopsy two tumours were found in the brain; one at the apex of the left frontal lobe. The second tumour lay in the mesial aspect of the right occipital lobe, projecting a few mm. above the level of the brain, firmly adherent to the pia and only slightly to the dura. Its length was 3 cm., height 3 cm., thickness 2.5 cm.—mostly buried in brain substance. It lay directly over the sulcus hippocampi, extending to either side of it. The base of the occipital lobe was not involved.

Féré reported the case of a female, aged fifty-two years, who in November, 1883, had a sudden apoplectic attack followed by right hemiplegia. She had partial and slight right hemianæsthesia to cold and pain. Hearing, taste and smell were normal; and typical right lateral hemianopsia, the vertical line passing through the point of fixation, was present. Autopsy showed a yellow patch destroying the greater part of the left cuneus and encroaching somewhat on the adjacent occipito-temporal convolution, the fifth temporal convolution of Ecker. No other lesions were present.

Seguin reports one personal case. The patient, a man forty-six years old, consulted him first in January, 1884, for insomnia and dyspepsia. He was treated for various symptoms with varying success. December 5th, 1884, he had an attack in which he complained of numbness in the left cheek, arm and face, and most marked in the hand and foot. He had no distinct hemiplegia, and no hemianæsthesia, but thought that tactile sensibility as in passing his fingers over objects was somewhat duller. He could not see objects on his turning his head and eyes, and testing revealed left lateral hemianopsia, with a vertical division line not including the point of fixation. Central vision was good. He continued with varying symptoms until May 17, 1885, when he died. During his long illness he had attacks of acute hallucinatory mania, both aural and hallucinatory illusions being present. He had chills, high fever, and sweats which followed no distinct periodicity. Previous to his death his speech was sometimes somewhat difficult to understand. The hands both showed disorders of movement, choreiform tremors, and in the left hand slight ataxia and larger motion. The hemianopsia persisted to the last unchanged. The central vision remained good. The brain lesion which doubtless caused the hemianopsia

was found on autopsy to be a large focus of yellow softening, evidently an old patch, involving the basal part of the cuneus, the fourth and fifth temporal convolutions of Ecker, and a part of the hippocampal gyre. Other lesions were present in the brain, but as this case was in harmony with others Seguin regards it very properly as of great value.

In his table in the paper referred to on cortex hemianopsia, Seguin has included five cases of traumatic hemianopsia due to injuries of the occipital region of the skull, and lesion of the subjacent brain. I will refer to only one of these cases, that of Keen and Thomson, which is of historical as well as of scientific interest. Through the courtesy of Dr. Keen, I have examined a cast of the head of this patient. The case was reported in the photographic *Review of Medicine and Surgery*, February, 1871; also in the 'Medical and Surgical History of the War of the Rebellion,' Part 1, p. 206-207. It is also referred to in Flint's 'Physiology of Man,' vol. v., p. 41-42, 1874, as one of the first cases to show that the filaments from the optic tracts on the two sides are connected with distinct portions of the retina. The patient was kept under observation off and on for a number of years, and was examined and exhibited by Dr. Seguin at the time of the reading of his paper. His hemianopsia was found unchanged twenty-three years after the reception of his injury. The patient died within a year or two, but unfortunately no autopsy was secured. He was hit on the head by a Minie ball at the battle of Antietam, September 17, 1862. The wound of entrance was in the middle line one and a quarter inch above the external occipital protuberance; it made its exit about two inches to the left of the middle line, and three above the wound of entrance. He noticed impairment of vision two days after the injury. About ten days afterwards he had an attack of loss of consciousness with some paralysis of the right arm and right leg, which lasted some two or three months. His memory was imperfect for some months, but he had no aphasia. In walking he was very giddy, and noise and laughter would hurt him. His mental and physical powers gradually grew better. He was seen and examined by Drs. Keen and Thomson, in December, 1870. Among other conditions he was found to have a complete lateral hemianopsia. Upon testing the field of vision it was found to be divided in each eye by a line passing through its centre in a vertical direction—total blindness existing to the right, and perfect vision to the left of this line. Ophthalmoscopic examination showed no pathological appearances. When Dr. Seguin

examined this man in 1885, he presented no distinct paralysis, no anæsthesia, no aphasic symptoms. His tongue deviated a little to the right, and the grasp of the right hand was a little lighter than that of the left. A rough test with a small white object at eighteen inches showed right lateral hemianopsia, with a line passing outside of point of fixation, and a darkened area in the left upper temporal quadrant. Pupillary reaction was normal. Examination of fundus showed blood vessels of normal size; outer temporal quadrant of each disc whiter than normal; left a little whiter than the right. An experiment on the cadaver showed that the track of the ball was such that it must have injured the optic fasciculus on its way to the cuneus.

The most important case which has been recorded since the paper of Seguin is that of Hun, in which a defect in the fields of vision involving the lower left quadrant of each eye occurred with atrophy of the lower half of the right cuneus.

The patient, a man aged fifty-seven, in 1869 had a severe attack of double pneumonia, and during the year following slight attacks of vertigo while walking, which were attributed to weakness. From 1877 until his death, he was troubled by slight deafness and by more or less roaring in his ears. In 1882 he had a large carbuncle on his neck, and after that time he seemed less vigorous than before. Early in December, 1884, he was examined by Dr. Merrill, who found normal reaction of the pupils, normal appearance of the fundus, vision and colour perception perfect, but a defect in the fields of vision involving almost the whole of the left lower and the peripheral portion of the left upper quadrants in each field. The defect was somewhat more extensive, especially as regards the upper quadrant, in the left field of vision than in the right.

From this time the condition of the patient did not change materially. He continued to be very nervous, and at times irritable and suspicious. He exhibited no paralysis of motion or sensation. His memory was weak in regard to names; he often called the same person by several names in the course of conversation; while in other respects, as in recognising faces, his memory was excellent. He slept but little, and in his sleep there was much twitching of the limbs. In 1885 he had a severe attack of angina pectoris, and for two weeks he could walk only a short distance without bringing on an attack of the pain. In February, 1886, he had his most severe attack of angina, lasting several hours, and from this attack until his death he was scarcely a day without pain in the precordia or in the arm.

During the last month of his life he vomited often. He complained of increasing dulness of vision and of greater angle of obliteration, and was much troubled by a new building near by appearing to be out of line. On May 7, 1886, while quietly walking in the street, he sank gently to the ground and died.

At a point on the median surface of the right occipital lobe was complete atrophy of the cerebral convolutions, only a trace of them remaining as a delicate gray gelatinous fringe. This atrophy was strictly limited to the lower half of the cuneus; being bounded below by the calcarine fissure, in front by the parieto-occipital and above by a curved line which started from the parieto-occipital fissure, and arching backwards across the middle of the cuneus terminated at the posterior border of the median surface close to the calcarine fissure. The white matter underneath the point of atrophy was softened to a depth of about one-third of an inch. There was no deposit of pigment in the neighbourhood. The corresponding point on the left occipital lobe showed no atrophy, nor did any of the other cerebral convolutions. Sections through the brain substance, the optic thalami, and the other ganglia at the base revealed nothing abnormal. The optic nerves and tracts showed no microscopic atrophy or degeneration. No microscopical examination was made.[1]

Views of Physiologists as to Visual Localisation.

The view of Ferrier as expressed in the latest edition of his 'Functions of the Brain,' is that the angular gyres maintain relations with the areas of clear vision, and as a matter of course especially with the macula luteæ.

This physiologist has modified his earlier views in so far that he no longer localises the visual centres in the angular gyres to the exclusion of the occipital lobe; but believes now that the visual centres embrace not only the angular gyres, but also the occipital lobes, which together he terms the occipito-angular regions.

Recently considerable activity has been exhibited in the investigation of visual areas.

From a long series of experiments upon the monkey's brain,

[1] At a recent meeting of the Philadelphia Pathological Society, I presented the brain of a man who had been blind more than twenty-five years—how much more could not be positively ascertained. Both occipital lobes were unquestionably small. The cuneus on each side was small, the first occipital convolution of Ecker (superior external *pli de passage* of Gratiolet, and par-occipital of Wilder) showing lack or arrest of development. The second and third occipital convolutions of Ecker, especially on the left, presented a narrow, dwindled appearance. In another brain of an old woman, blind for at least thirty years, similar gross appearances of arrested development in the occipital region were present.

on which he was engaged with Mr. Victor Horsley during more than two years, Schäfer[1] writes as follows:

"With regard to vision our experiments were not conclusive. We found that extensive lesions, both of the occipital lobe and of the temporal lobe, were invariably followed by visual disturbances, taking the form, when the operation was confined to one side of the brain, of bilateral homonymous hemianopsia; but in nearly every case the hemianopsia was merely temporal, and after a certain time we could not in our monkeys obtain any distinct evidence of the persistence of the visual defect. The most marked results of this kind were obtained when the occipital lobes were the seat of operation, extensive unilateral lesions in this region producing hemiopia, and bilateral lesions producing amblyopia; but in neither case were the symptoms permanent, and after a time the animals, so far as we were able to determine, could see as well as their intact fellows. In one case only did the hemiopia persist, and this was one in which, after a bilateral lesion of both occipital lobes had been carried out and the temporary blindness thereby produced had been recovered from, the angular gyrus of one side was destroyed. This second operation, made upon the animal in which the occipitals had already been extensively destroyed *without* permanent blindness, did produce a condition of hemianopsia which lasted until the animal's death some three months later. We were of opinion at the time that this instance might warrant us in taking up a position similar to that of Luciani and Tamburini, and intermediate between those of Ferrier and Munk—the former of whom originally denied the participation of the occipital lobe in the visual perceptive function, and still appears to regard it as subordinate to the angular gyrus; whereas the latter would localise those perceptions entirely in the occipital lobe and deny all participation of the angular gyrus. But we made only four experiments upon these regions, and in none of them was the removal of the occipital lobe complete, as was proved by *post-mortem* examinations of the brains. They were not, therefore, decisive against Munk's statement, that persistent hemiopia or blindness follows extirpation of one or both occipital lobes alone, and it became necessary to pursue further inquiries in order to test its accuracy."

In conjunction with Dr. Sanger-Brown, Schäfer also experimented upon the angular gyre and the occipital lobes. Destroying one angular gyrus as completely as possible with the actual cautery they could discover no defect of vision, no loss of movement of the eyes or eyelids, and no anæsthesia of the corneal conjunctiva. A week later the angular gyre of the opposite side was destroyed, also with negative results.

[1] *Brain*, London, January, 1888.

In illustration of the effects produced by complete removal of the occipital lobe, and of that alone Schäfer gives two instances, in one of which the operation was unilateral, in the other bilateral.

"In the monkey upon which the unilateral operation was performed, the left occipital lobe was removed by a vertical incision carried along the line of the parieto-occipital fissure. That the removal was exact and complete was confirmed on *post-mortem* examination, some eight months after the establishment of the lesion, when it was seen that the whole of the occipital lobe, and only this lobe was involved, the angular gyrus being quite intact and normal, and the surface of the section looking as fresh, and showing as clearly the distinction of grey and white matter, as if the operation had just been performed. The result was the immediate establishment of bilateral homonymous hemianopsia, which persisted the whole time the monkey was kept alive. Objects so placed that their images fell upon the left half of the retinæ were taken no notice of: a threatened blow coming from the right-hand side of the mesial visual plane was winced at or avoided; currants strewn upon the floor were only picked up towards the left side, the animal working in that direction. In the case of the monkey with bilateral operation the result was total and persistent blindness."

The views of Hun [1] are that the convex surface of the occipital lobe, particularly of the left side, is associated with complete visual perception and recognition; and also that the so-called left angular gyre is essential for the memory of the appearance of words, lesions of it causing alexia and agraphia.

I shall not attempt to analyse, criticise, or reconcile these various and varying views. They agree at least in showing the production of hemianopsia from lesions of the cuneus and adjacent occipital lobe.

The general visual zone which has been determined can probably be compared as to subdivision with the general motor zone. As the motor zone has been subdivided into areas of representation, not only for the leg, trunk, arm, face and speech, &c., but also into areas or centres for parts of the leg, arm, face and speech, so efforts partly successful are now being made to subdivide the visual zone. The retina, so far as its connection with the central cortex is concerned, can be subdivided into segments probably of a somewhat regular shape. At first, studies in hemiopia and hemianopsia seemed to show that the only definite connection was between the halves of the retinæ and cortical centres, but the latest observations indicate that quad-

[1] *Op. Cit.*

rants, and probably even smaller portions of the retinal expansion are related to separate areas in the brain. The macular region almost certainly has its special cortical centre. The tendency of investigation, both experimental and pathological, is to show that the whole of the occipital lobe, and adjoining portions of the parietal and temporal lobes, are in some way concerned with vision, as I have tried to indicate in the diagram.

With reference to visual localisation, the importance of confirming physiological experimentation by careful clinico-pathological observations cannot be over-estimated; these observations are here even of more importance than in motor localisation. The movements of a limb or a portion of a limb can be studied with considerable accuracy in the lower animals as well as in man: but in investigating sight, the other special senses, or general sensibility, in the lower animals, we are confronted with special difficulties and sources of error. This is perhaps more strikingly true of hearing, taste and smell than of the other senses, but it is true for all. Schäfer, referring to a monkey from which he believed he had eradicated both angular gyres, states that for three or four weeks the animal failed to see objects which were just below or to one side of its eyes, and even at the time of writing there appeared to be complete absence of vision in the antero-superior and lateral portions of the retinæ; but it was difficult to prove this, because the monkey had acquired the habit of rapidly directing his head and eyes so as to use the central parts of the retinæ.

Operations guided by Visual Localisation.

In spite of the conflict of views with reference to visual localisation, sufficient has been determined to indicate several positions for operation guided chiefly by visual symptoms.

If a patient has lateral homonymous hemianopsia as the special localising symptom, operation should be performed with the view of reaching the cuneus behind the position of the parieto-occipital fissure. If, without hemianopsia, the patient fails in intelligent recognition of things and words, the aim should be the lateral occipital convolutions and the angular gyre, which is adjacent, or indeed may be situated in the occipital lobe. When, with lateral homonymous hemianopsia, the patient also has hemianæsthesia, the lesion is probably in the tracts between the cuneus and the primary optic centres, large enough also to involve the sensory tracts. Such a lesion would probably best be reached

beneath the position where, on the lateral aspect of the hemisphere, the parietal, occipital and temporal lobes come together. Hemianopsia is sometimes a late symptom, the result of invasion of the visual region from other localities; such a case will be referred to later when speaking of large lesions of the temporal obe.

I have made various examinations of the human brain on recent as well as older specimens in order to determine, if possible, where the optic radiation proceeding from the corpora geniculata and corpora quadrigemina enter the hemisphere to proceed to the cortical centre of vision; in part with a view of determining the relations of the angular gyrus, cuneus, and adjoining occipito-temporal region to this place of entrance, and therefore the position and direction of the intra-cerebral tracts. Undoubtedly, so far as the human brain is concerned, both the angular gyrus and the occipital lobe are so placed with reference to the geniculate bodies and corpora quadrigemina that they are anatomically and morphologically in relation with these radiations. Schäfer [1] gives a view of a longitudinal section of the hemisphere of a monkey which he believes demonstrates that fibres proceeding to the angular gyrus are not involved in lesions of the occipito-temporal regions. Whatever may be true of the monkey, this is certainly not true of the human brain.

Wernicke's Hemiopic Pupillary Inaction.

The great practical importance of some of the studies which have grown out of cerebral localisation is seen in the fact that the simple but valuable discovery in semiology, Wernicke's hemiopic pupillary reaction, enables us with a flash of the ophthalmoscope to throw the lesion producing hemianopsia back of the primary optic centres. With a normal retina uniform pupillary reaction occurs, no matter at what position the ray of light strikes upon it. Seguin [2] clearly explains this hemiopic pupillary reaction, or inaction, as he suggests to call it, in one of his papers on hemianopsia. In a word, the test depends upon the fact that the hemiopic part of each retina being physiologically inert, fails to receive any impulse from the light which is thrown on it. When the lesion is in the cuneus or occipital lobes, hemiopic pupillary reaction is not observed. When the lesion producing hemianopsia involves the optic tracts, there is from an early period hemiopic pupillary inaction as well as partial nerve atrophy. A full dis-

[1] *Brain*, July, 1888.

[2] *Jour. of Nerv. and Ment. Dis.*, vol. xiv., November and December, 1887, 721-737, and *Fortschritte der Medicin*, I., Heft 2, 1883, cited by Seguin.

cussion of this subject will be found in the paper of Seguin, and the original memoir of Wernicke.

Morphological Peculiarities of the Human Brain.

It is a question whether we are in a position for practical purposes to make any distinction between the angular gyre and the external occipital convolutions; nor is it absolutely necessary that this should be done in trephining with a large instrument.

Morphological peculiarities of the human brain have an important bearing on this subject. In 1880,[1] I called attention to this matter and particularly with reference to the so-called angular gyre, referring to the brain of a white man in which the first temporal sulcus extended back of the transverse occipital and to within half an inch of the longitudinal fissure. The angular gyre is usually regarded as the arch composed of the posterior uniting portions of the inferior parietal and superior temporal gyres. It has seemed to me that these morphological aberrations are especially marked in the parieto-occipital region, so much so in some cases as to lead us to use great care in determining by external methods the position of the cerebral areas.

It will only be necessary to call attention to one or two striking peculiarities in the brain examined, mostly in the insane and in individuals of low organisation.[2] In a case of delusional monomania, for example, the occipital fissure showed itself and in its communications and surroundings, marked ape-like conditions, such as might have been very deceptive if exposed by trephining.

In the other hemisphere of the same case the cuneus was small, and the first temporal fissure presented an unusual appearance, being divided about its middle by a comparatively large gyre. In another case, in the right hemisphere, the second temporal fissure was short, and communicated with a very well defined almost vertical fissure, which separated the temporal from the occipital lobe. The first temporal, as well as the second, communicated with this fissure, the vertical extension of the first being, in fact, continuous with it. The cuneus was distinctly smaller and of a different shape from the cuneus of the right hemisphere. In another case, the first temporal fissure was remarkable in that it was completely confluent with the Sylvian, extending from the point of the confluence as a deep, well defined

[1] *Phila. Med. Times*, April 24, 1880, 366-370.

[2] *Jour. Nerv. and Ment. Dis.*, vol. xii., September and October, 1886, 517-553.

trench backwards and upwards nearly to the sagittal fissure, crossing and confluent with the interparietal. In a Chinese brain studied by Dr. A. J. Parker and myself, a remarkable peculiarity of the brain was that in the right hemisphere the first temporal fissure, beginning near the anterior extremity of the temporal lobe, passed backward across the entire extent of the parietal lobe and over the median edge of the hemisphere for the distance of half an inch, terminating in the precuneus just in front of the parieto-occipital fissure.

This is an interesting field, and has some practical importance. Even the Sylvian, the central, and the parieto-occipital fissures have considerable variations, the first in its horizontal level and extension, and the others in their antero-posterior position. I have reported one case in which the Sylvian fissure took a position much more vertical than usual and reached nearly to the longitudinal fissure. As a rule, variations, such as those referred to here, for fissures like the Sylvian, would not confuse us in operating after the methods of Broca, Thane, Reid, Horsley, and others; but they are certainly worthy of some consideration.

Localisation of Cutaneous Sensations.

With reference to the localisation of the centres for cutaneous sensations—touch, pain, temperature, &c.—we are acquiring more positive knowledge and may be able in time to use such knowledge to guide in surgical operations. The evidence, physiological, clinical, and pathological is becoming stronger in favour of the existence of a zone for these general sensations separate from the motor areas.

Various, indeed, have been the views held with reference to the localisation of these sensations. Luys, largely from anatomical studies, made the thalamus a centre of sensibility, subdividing it into four special centres—olfactory, optic, acoustic, and for general sensibility. Ferrier regards the thalamus as in some way connected with sensation, but believes that differentiated areas for sensation also exist in the cortex. He holds that it is probable that the thalami are especially related to the sensory tracts, and the striate bodies to the motor tracts; that these ganglia represent in a subordinate manner all the sensory and motor tracts of the cortex. The thalamus is a centre of conveyance, or an interrupting ganglion in the course of the sensory tracts. The experiments of Monakow led to the conclusion that different portions of the thalamus are related to

cortical areas. Fournier believes that sensory fibres terminate in the thalamus. Crichton-Browne regards this ganglion as a great centre of general sensibility: but Flourens, Longet, Schiff, and Tamburini, among others, have assigned to it motor functions. The weight of the evidence, both from experiment and disease, connects the thalamus with various forms of sensation. It is probably a halting place where sensory impressions going from the periphery to the cortex undergo some transformation. It is necessary, at least, briefly to thus refer the functions of the thalamus as some authorities are inclined to regard it as the sole cerebral region for sensibility.

From analogy and general principles it is altogether probable that we have above the ganglia a separate localisation for the different forms of cutaneous sensibility. As Ferrier puts it, up to the point of radiation towards the cerebral cortex the sensory paths have been proved to be entirely differentiated from the motor, and it is therefore extremely unlikely that the two should become jumbled together indiscriminately in the cortical centres. We have not only the well known differentiation between sensory and motor nerve roots and tracts in the spinal cord, oblongata, pons, and crus cerebri; but abundant evidence as to the separation of sensory from motor tracts in the internal capsule. The very simple but natural question may be asked: where do these paths lead if not to special cortical areas for sensation? Cases have been reported by Demeaux (quoted by Ferrier), by F. Müller and others, in which autopsies have shown hemianæsthesia to result from lesion of the hinder third of the internal capsule or adjoining portions of the corona radiata.

Veyssière produced hemianæsthesia on the opposite side of the body by dividing this portion of the capsule in animals. We not infrequently see hemiplegia and hemianæsthesia united in the same patient as the result of a capsular lesion, and numerous autopsies have shown that the posterior and middle parts of the capsule are in these cases involved in a destructive lesion. Cases of hemianæsthesia from lesions both of the thalamus and lenticular nucleus have been reported, but in very few has the lesion been strictly localised to these ganglia, and even in these it is a question whether the hemianæsthesia resulting has not been due to pressure on the internal capsule.

Recently I have made a *post-mortem* examination on a patient who died at the Philadelphia Hospital, a woman forty-seven years old. She had had many symptoms which were supposed to be hysterical. On the morning after her admission she

had a fit or seizure, the character of which was not closely observed. Afterwards, however, she was paralysed and anæsthetic over the right half of the body and limbs, her speech also being thick, but consciousness not lost. Partial anæsthesia was present over the left half of the body. Knee-jerk was increased, and ankle clonus was present on both sides. She improved rapidly, but remained in bed two weeks. Two days before her death she had an apoplectic attack, and became completely paralysed and hemianæsthetic in the face, limbs, and trunk of the left side. The autopsy showed a foyer of softening in the right hemisphere, including the entire breadth of the internal capsule, two-thirds of the adjoining lenticular nucleus, a small anterior external portion of the thalamus, and a narrow strip of the caudate nucleus where it curves around the thalamus. Careful transverse sections showed that the anterior boundary of this focus of softening was one and a-quarter inches behind the head of the caudate and lenticular bodies, and that it extended from backwards about five-eighths of an inch. It was so situated that a transverse section immediately in front of the thalamus marked its anterior boundary. The softening of the narrowed caudate nucleus was all that was observable in opening the ventricle. The left hemiplegia and hemianæsthesia might very properly be attributed to the complete transverse destruction of the internal capsule. A large recent embolus was found in the right middle cerebral artery. It is difficult to account for the temporary right hemiplegia and hemianæsthesia, except on the theory of hysteria or functional disturbance, as no lesion of the cerebral hemisphere on the left side was discovered.

The fibres of the sensory tract are probably separate from the motor even in the centrum ovale.

"The third set of fibres of the projection system," says Starr,[1] "includes those which lie just posterior to the motor tract, and which pass inward from the parietal convolutions. These take a similar course to the motor tract, and fill up to a considerable extent the space between it and the radiation of the visual tract, towards the occipital lobe. They are mingled with fibres which pass to the optic thalamus, but are separable from them, as Edinger has shown, in fœtal brains, and may be traced down through the capsule to the tegmentum of the crus, where they divide into a portion going to the leminscus, and a portion going to the formatio reticularis. This set of fibres conveys the

[1] *Medical Record*, Feb. 13, 1886.

sensations of touch, pain, temperature, and muscular sense, and lesions in its course will cause disturbance of these sensations. Like lesions in the motor tract, the rule obtains that the nearer the cortex the more likely is the lesion to cause an affection of a single limb, while the nearer the capsule the more likely is the symptom produced to be hemianæsthesia."

Ferrier in his earlier experiments found that lesions involving the horn of ammon and hippocampal convolution, caused impairment or abolition of tactile sensibility on the opposite side of the body, and located the cortical centres for this form of sensibility in the hippocampal region. In experiments with Professor Yeo, in 1884, he established lesions in the hippocampal regions in ten monkeys, and in five of these in both hemispheres, and these lesions showed that tactile sensibility was in every case impaired or abolished, according to the amount of destruction of the hippocampal and temporo-sphenoidal region. It was also established, however, that a very extensive lesion might be made in one or both hippocampal regions without producing permanent anæsthesia.

Munk [1] claims that the area of common sensation—including sensation of pressure, location of a limb, muscular sense, and touch—lies in the central region, including the anterior and posterior convolutions and adjacent cortex. He divides this area into special centres for the hind leg, fore leg, head, eye and ear muscles, neck and body; holding that these coincide with but are somewhat more extensive than the corresponding motor centres for these parts.

The sensations of touch, according to Luciani, are perceived in the central convolutions, and therefore lesion of these cause anæsthesia as well as paralysis. The tactile area includes the parietal convolutions also, but does not reach the occipital and temporal lobes. This author holds that all sensations appear to have a common zone in the parietal region, and lesions in this common zone may cause disturbance of all the senses.

It is worthy of remark that the physiologists and others, who contend that the sensory portion of the cortex coincides with the motor, also hold that it extends beyond into adjacent cortical regions, as witness the views of Munk, Tripier, Moeli, and others cited by Starr in his elaborate paper.

Ferrier's explanations [2] of what he regards as the errors of

[1] *Pflüger's Arch. f. Physiologie.* Quoted by Starr, *Jour. Nerv. and Ment. Dis.*, vol. xi., No. 3, July, 1884, 327-407.

[2] 'Functions of the Brain.'

observers like Schiff, Munk and others who believe that sensory areas coincide with motor centres, is reasonable. "The conclusion that tactile sensibility is lost or diminished after the destruction of the cortical motor area," he says, "is based on defective methods of investigation and erroneous interpretation of the reactions of the lower animals to sensory stimulation. Though an animal does not react so readily to sensory stimulation of the paralysed side it does not follow that this is due to diminished or absent perception of the stimulus. An animal may not react, or react less energetically, to a sensory stimulus, not because it does not feel it the less, but because it is unable or less able, to do so from motor defect. It is astonishing what apathy or indifference some animals display towards certain forms of stimulation, such as gradually increasing pressure on the fingers or toes which one would regard as well calculated to elicit reaction or signs of uneasiness. Unless the stimulus is of a nature to at once excite attention, or to evoke reflex action, it may appear to be altogether unperceived. All that the experiments of Schiff and Tripier demonstrate is that motor reactions are less readily evoked on the side opposite the cortical lesion. But the same thing occurs in cases of purely motor hemiplegia in man."

On anatomical and morphological grounds Broca holds that the callosal and hippocampal regions constitute together a distinct lobe of the brain, the falciform or limbic lobe; also I believe erroneously, that this lobe is connected entirely with the sense of smell. So far as the brain of man is concerned, in this lobe should probably be included the entire precuneus or quadrate lobule on the mesial aspect of the hemisphere and the adjoining portion of the superior parietal convolution on the lateral aspect of the hemisphere.

The most important recent contribution to cortical sensory areas is certainly that of Horsley and Schäfer.[1] Hemianæsthesia partial or complete and contra-lateral resulted from destructive lesions of the limbic lobe. They found that any extensive lesion of the gyrus fornicatus was followed by hemianæsthesia more or less marked or persistent; sometimes the loss of sensation involved almost the whole of the opposite side of the body; sometimes it was localised either to the upper or lower limb, or to a particular part of the trunk. They did not, however, succeed in establishing the relations between special regions of the body and the parts of the convolutions which had been destroyed.

"These experiments were frequently, but by no means in every

[1] Phil. Trans. Royal Soc., 1888.

case, complicated by the presence of a certain amount of motor paralysis, chiefly, if not entirely, affecting the muscles of the leg. We have no doubt that this condition was always due to a lesion (accidentally produced during the operation, or subsequently as the result of interference with the circulation) in the leg area of the marginal convolution. Now in one or two of these cases the anæsthesia affected chiefly the paresed limb, and it might be argued by those who, like Schiff and Munk, hold that the excitable areas of the cortex are concerned with the perceptions of sensory impressions from the corresponding regions of the body that the loss of sensibility was due to lesion of the motor area. But against this argument we may not only put forward those experiments in which there has been no accompanying paralysis, but also others in which the hemianæsthesia has been well marked in the upper limb and upper part of the trunk, while the lower (paresed) limb has exhibited no diminution of sensibility whatever. . . . .

"From which it would appear that while the whole limbic lobe may be concerned in the perception of sensory impressions, the part played by the gyrus fornicatus, at least as regards cutaneous sensibility, is more important than that played by the hippocampal portion of the lobe. But until it has been possible entirely to destroy the whole lobe upon both sides of the brain it is impossible to determine whether it is assisted in its functions by any other portion of the cerebral cortex. . . . .

"The results of our experiments upon the limbic lobe seem to point to the conclusion that this portion of the cerebral cortex is largely, if not exclusively, concerned in the appreciation of sensations, painful and tactile. This is an extension of the view put forward by Ferrier, who was inclined as the result of his own experiments to limit that function to the hippocampal region. Dr. Ferrier was good enough to assist at some of our experiments upon this part, and has fully accepted the conclusions to which they point."

Starr, from a series of American cases of cortical lesions of the brain,[1] and also from a study of the sensory tracts,[2] concluded that the various sensory areas lie about and coincide to some extent with the various motor areas for similar parts; that, in other words, the Rolandic region is a sensory-motor region, the sensory area, however, including to some extent the gyres of the adjacent postero-parietal lobe. Collections of cases such as

[1] *Am. Jour. Med. Sci.*, Phila., 1884, n. s., lxxxvii., 366-391.
[2] *Jour. Nerv. & Ment. Dis.*, vol. xi., No. 3, July, 1884, 327-407.

these cannot, however, overcome positive evidence of decided destructive lesions of the cortical motor centres, without any disturbance of touch, pain or temperature, or even of the muscular sense; and a close study of the cases of cortical lesion in which both motor and decided sensory phenomena have been present will not bear out fully the view of Starr.

One of Starr's tables contains forty-one cases showing cortical lesions of the brain with sensory symptoms. That the sensory area lies about the motor region might even from these cases be conceded, but that the two coincide has been by no means proved, particularly if the postero-parietal region is not regarded as a necessary part of the motor area. Reviewing the forty-one cases, in only one-fifth to one-sixth are the lesions restricted to parts of the brain in front of the central fissure, although in many of the others both the anterior central and posterior central gyres are involved. In more than thirty of these cases the reported lesions involved in part or whole the postero-parietal region and posterior central gyre. Four of my cases are included in the table, and this has led me to look over not only these but also other of my cases of cortical lesion with sensory phenomena; and from doing this the conclusion has been reached that they do not support the doctrine that sensory and motor areas entirely coincide.

In the case of a fibroma which involved the first and second frontal gyres and anterior segment of the gyrus fornicatus and corpus callosum, anæsthesia of the conjunctiva, with conjunctivitis and corneitis were present. In a patient with a gumma involving the upper fourth of the pre-central and a smaller segment of the post-central gyre, hyperæsthesia was marked, with also crural Jacksonian epilepsy, but the lesion grazed the sensory area. A carcinoma of the middle portion of the posterior central gyre and the upper part of the inferior parietal lobule was accompanied with wide-spread destruction of the corona radiata, and impaired sensibility of the limbs of the opposite side with other phenomena was present. Owing to the posterior extension of this growth and the large sub-cortical destruction, this lesion could certainly not be claimed to teach anything with reference to the coincidence of motor and sensory areas. In another case of tubercular tumour in the motor area, the interior of the hemisphere was broken down, and hemianæsthesia, at first partial but later complete and persistent, was present.

Numerous observations have shown me that hyperæsthesia is a common phenomenon in cases of diffused cortical tubercular meningitis, and this hyperæsthesia may involve any or all the

extremities or the face. This fact is not to be explained as simply from irritation of the pia mater; for on the other hand I have observed cases of localised lepto-meningitis in which both hyperæsthesia and even pain in the head were absent. Such cases also cannot be fully explained by tubercular deposit in and inflammation of the dura mater. Although dural meningitis may cause headache of great severity and sensory phenomena in the domain of the fifth nerve, it should not be held responsible for hyperæsthesia of the extremities. The hyperæsthesia in these cases is best explained by irritation of sensory cortical areas.

Bernhardt[1] has recorded the case of a woman, forty-one years of age, who after an apoplectiform attack had hyperæsthesia of both lower extremities; also paresis of the right arm, lower face and foot; spasm involving the right hand, arm and face; weak reflexes of the right leg; confusion, excitability and aphasia. The lesions were a tubercular growth in the upper extremity, lateral and median portion of the left posterior central convolution and in the precuneus; a similar growth in the middle portion of the posterior central gyre; and a tubercle the size of a pea at the anterior extremity of the corpus striatum. The hyperæsthesia in the lower extremities might be explained by the tumour in the great median fissure. Hitzig[2] quotes from Löffler the account of a soldier who received a gunshot wound on the top of the head, the dura mater being pressed inward by the depressed bone at a point which corresponded with the upper ends of both central gyres. Both legs were hyperæsthetic and also paralysed. Such symptoms can be explained by pressure on the paracentral gyres of both sides and direct or inflammatory irritation of the neighbouring sensory cortex below or behind.

Bramwell[3] has reported a case of incomplete paralysis of the left extremities and of the face, with disturbance of sensation of both the arm and leg; with also atrophy of both optic nerves and other sight disturbances. The autopsy revealed a tumour with softening of the surrounding brain in the right superior and middle parietal convolutions and a part of the first occipital convolution.

Brill[4] records the history of a patient with right-sided anæsthesia and paresis, the former more marked than the latter. In a few weeks hyperæsthesia developed, with also restlessness,

[1] *Arch. f. Psych.*, iv., heft 3, 1874.
[2] *Op. Cit.*
[3] *Lancet*, September 4, 1875, ii., 346.
[4] *N. Y. Med.* vol. xxii., 1882, July 15, 81-82.

tinnitus, beclouded mind, slight amnesic aphasia and colour blindness, the patient not being able to tell green from red, although his field of vision was not limited. A triangular focus of softening was found in the left cuneus, most of the cuneus being involved.

Fortunately I had the advantage a few hours before delivering this address of hearing one of the best papers yet written on the cortical localisation of cutaneous sensations, read by Dr. C. L. Dana before the American Neurological Association, and since published in the Journal of Nervous and Mental Disease for October, 1888. Dr. Dana discusses the three views with reference to this localisation: (1) that the centres for these senses are entirely separate from motor areas; (2) that the cutaneous sensory centres are more or less identified with the motor centres; and (3) that both the motor zone and the limbic lobe are concerned in the representation of cutaneous sensation. His paper deals principally with pathological and clinical data. Altogether he collected 142 cases, including four personal observations. He concludes, among other things, that the clinical and pathological evidence collected by him shows that the motor areas of the cortex contain also the representation of cutaneous sensations; and that cutaneous anæsthesia of organic central origin is always limited to or more pronounced in certain parts. The same objections apply to most of his cases as indicated for those of Starr.

Dana collected for this paper twenty cases in which the gyrus fornicatus or hippocampal convolution were more or less involved, and in none of these, he says, was any anæsthesia present that could be fairly attributed to the lesion. Even a casual study of these cases seems to me to show their weakness for the support of the position taken that the limbic or falciform lobe is not concerned with sensation. In nearly one-third of these cases some disturbance of sensation was present. Three of the remaining cases were demented, and presumably any investigation into cutaneous sensation would have but little value; one case presented no symptoms.

What Ferrier says of defective methods of investigation and erroneous interpretation of the reaction of the lower animals to sensory stimulation may be applied with some additions to the study of cutaneous sensation in cases of cerebral disease in man. Numerous sources of error are present, not only in investigating but also in reporting the results of investigation.

To test cutaneous sensibility requires time, patience, and method. It is often tedious and monotonous; but it must be done

carefully or the results, particularly for tactile sensibility, are worthless. Patients cannot often be depended upon, as with reference to such tests the greatest differences in the personal equation are found. Some patients have a fashion of stopping to think or weigh their answers when being tested for cutaneous sensation. This often renders the answers worthless; the response should be given promptly.

One of the commonest methods of investigating cutaneous sensibility is that of Weber, which depends upon the fact, or the alleged fact, that the distance apart at which two points can be discriminated is much the same for the same local areas in different individuals. Tables have been published which are supposed to show the least normal distances at which two points can be distinguished. This method, if very carefully applied, is sufficiently correct for many cases, but often the results obtained from such testing are utterly worthless. I have known not only patients with real or alleged loss of sensation, but perfectly healthy individuals to give diverse and confusing answers when tested with the æsthesiometer. Some of the records of the presence of anæsthesia or analgesia in reported cases have doubtless been made improperly as the result of carelessly testing the power of the patients to discriminate between two blunt or sharp points.

Another source of error, particularly in recording cases, is in deciding that objective insensibility exists from the statements of the patients with reference to their feelings. Whatever may be the explanation, it is true that many, though by no means all patients suffering from motor paralysis the result of a cortical or sub-cortical lesion, complain of certain paræsthesias. Spontaneously or in response to questioning they will refer to a paralysed member, or a portion of it, as feeling heavy or numb, or cold, or as if asleep; but often in these cases careful and elaborate testing for true depression or loss of sensation will fail to reveal its presence in the slightest degree. I have been for some time engaged in minutely studying cases of hemiplegia, monoplegia, and aphasia with a view of determining in the most detailed manner the exact movements or forms of speech impaired or abolished, and also whether or not in these cases localised defects of sensation were present. The investigation is a clinical study in localisation, the value of which will be much enhanced by the number of autopsies obtained in the cases studied, but even without such autopsies some results both curious and valuable can be obtained. A single case will serve to illustrate the point which I wish to make about some sensory investigations

and records. A young man, with a history of rheumatism and several heart murmurs, was admitted to the Philadelphia Hospital three weeks after a sudden attack of right-sided hemiplegia with aphasia. On admission he had recovered almost entirely from the paralysis of the arm and leg, but decided facial paralysis and slight aphasia, or rather ora-lingual paresis, were still present. His motor symptoms were studied with minuteness and will be published in a paper in course of preparation. On inquiring as to sensory disturbance he said that he had had for several days some pain in the leg and arm, and a feeling of numbness or as if the parts were asleep in the right face, arm and leg. This sensation had disappeared entirely from the leg and arm with the abatement of the motor paralysis, but he still complained of decided numbness in the right cheek. The most careful tests were made as to the senses of touch, pain, and temperature, but not the slightest loss could be determined. He was examined by gently touching the cheek with the finger and a feather, with blunt and sharp points, with hot and cold water, with different weights and different amounts of pressure, and by the method of Oppenheim, namely, that of touching symmetrical spots on both sides of the median line at once, and observing whether he appreciated the touch on both sides equally.[1]

Some light has been thrown on the disputed question of the existence of sensory centres in the motor cortex by careful examinations of patients after operations, particularly when definitely determined gyral areas have been cleanly excised. In one of Horsley's cases, in which the scar tissue and surrounding healthy brain substance was excised, after the operation the patient had at first, coupled with some motor paralysis, a loss of tactile sensibility over the dorsum of the two distal phalanges of the fingers, and could not tell the position of any of the joints of the fingers, thus showing apparently some loss of tactile and muscular sense; but as Horsley remarked it was possible that some of the fibres of the corona radiata coming from the gyrus fornicatus may have been injured. Both motor and sensory paralysis disappeared in the course of two months.

[1] The patient recently died. At the autopsy, a focus of strictly cortical yellowish softening was found involving the lower extremities of the central convolutions both on their external and Sylvian surfaces, and a spot one-half an inch in diameter about the middle of the internal portion of the island of Reil. The softening reached into the fissure of Rolando, and also into the precentral fissure, taking in a posterior, inferior strip of the second frontal convolution. Its greatest height was 1½ inches upward from the Sylvian fissure, its width along this fissure was 1¼ inches. The lesion did not reach to the anterior branch of the Sylvian fissure, its anterior limit being one-fourth of an inch behind this fissure.

In Weir and Seguin's case of right-sided Jacksonian epilepsy and paresis—in which a small sarcomatus growth was cut out of the white substance below the posterior edge of the second frontal and the anterior edge of the pre-central gyres—careful tests were made over a period of nearly seven months after the operation. They showed at first slight apparent dulness of tactile sensibility in some parts; retention of pain, and temperature and muscular sense, with motor paresis and speech defect. In later tests the patient no longer felt numb; could tell the lightest touch on the fingers and hand; and with eyes closed could distinguish consecutive contacts with a coarse bed cover, a thin handkerchief, and a sheet of paper; he could also recognise slight difference of weight. Nearly seven months after the operation co-ordination of the hand was perfect; the patellar reflex normal and equal on both sides; there was no wrist reflex; the senses of touch, temperature normal; he could distinguish differences of weight of only a few grains, and was fully conscious of all passive movements.

In the case of Lloyd and Deaver, also one of brachio-facial spasm and paresis, reported at the meeting of the American Neurological Association during the session of the Congress, no gross lesion was found, and the facial and arm centres were carefully excised, with a resulting cure of the spasms. On several occasions, with Dr. Lloyd, I tested the conditions as to motor power, sensibility, and the reflexes in this patient, with results which have been reported by Dr. Lloyd. This patient blindfolded could instantly recognise the slightest touch on all points of the affected side; even light breathing upon his hand was at once detected; pain and temperature sense were normal; and he could discriminate between weights. If objects were placed in his paretic hand he often failed to recognise what they were, but apparently because he was not able easily to grasp and run his fingers over them and thus take in their form and bulk.

These experiments, which should be repeated with the greatest care whenever opportunity offers, seem to uphold the doctrine of pure psycho-motor centres rather than the sensory, motor or mixed theory. They certainly are not in accord with the view that the motor zones contain centres of the muscular sense; or that they are areas for pathic or thermic sensibility, or places of confluence of excitations, or centres acted on at a distance by inhibition. The temporary disturbances in sensation may be due sometimes to destruction of association fibres between related sensory and motor areas.

Bechterew[1] maintains with reason that the loss of sensation in animals who have had the motor area of the convolutions destroyed is apparent and not real; that they cannot withdraw the irritated extremity, though they feel the pain, because they have no control of the muscles. He also considers the loss of the muscle sense only apparent, because if the animal's paws be placed in an uncomfortable position its failure to be removed is due to motor inability rather than impaired muscle sense. The animal will move away if irritated on the affected limb, thus showing that it feels but cannot withdraw the limb except by moving the entire body. He concludes that tactile sensation is behind and external to the motor area, and the centre for muscle sense and pain is at the beginning of the fossa of Sylvius.

In several cases of surgical operation on lesions of the motor zone, it has been claimed that the presence of sensory symptoms supports the opinion that the motor areas subserve a sensory as well as a motor function; the case of Macewen,[2] for example, of proto-spasm of the hallux preceded by sensory impressions and followed by paralysis, in which a tubercular nodule was found in the upper part of the post-central gyre. Such a case may prove contiguity but not coincidence of motor and sensory areas.

The neurologist and surgeon must therefore depend on motor symptoms alone in fixing the site for operation in cases in which motor symptoms are definite. When positive sensory symptoms are present, they should regard these as indicative of the extension of the lesion towards either the limbic lobe or the posterior parietal convolutions; or the involvement of the fibres going or coming from these gyres in the corona radiata.

We have no clinical or pathological data with reference to the so-called muscular sense of positive practical value to the physician or surgeon. The hypothesis that the cortical motor zone is rather a zone for muscular sense has little to support it either in physiology or in clinico-pathology. I can see no reason for requiring a muscular sense entirely distinct from other acknowledged forms of sensibility. Some facts have been brought forward which are supposed to indicate that a separate cortical area for registering impressions of muscular sense exists in the parietal lobe behind the motor zone, probably in the inferior parietal lobule; but these observations are not convincing; they certainly cannot yet be made of value in topographical diagnosis for surgical purposes.

[1] *Neurol. Centralbl.*, Leipz., 1882, ii., 409-414.
[2] *Lancet*, Lond., August 11, 1888.

The conclusion is warranted that there is a region for general sensation, including touch, pain, temperature, and possibly pressure, and location of a limb, which can be divided into special sub-areas for the various distinct portions of the body, and that these regions lie along side of and have close anatomical and morphological relations with corresponding motor areas, but that they are not identical with them. From an anatomical and morphological point of view, and from the facts of physiology and pathology, no part of the brain is more likely to contain these differentiated areas for sensation than the gyrus fornicatus, the hippocampal gyre, the precuneus, and the postero-parietal convolutions.

The Pre-frontal Lobe and the Cerebellum.

For the pre-frontal lobe and the cerebellum our diagnostic guides are gradually becoming more definite. We may not be able with the same absolute confidence from positive symptoms and signs to indicate lesions in these localities as we can point to the motor and visual zones, but we often can make the topographical diagnosis with sufficient certainty even for surgical purposes by combining various modes of investigation, as: (1) by a few positive localising symptoms; (2) by the general symptoms —such as, in brain tumour, for instance, choked disc, secondary atrophy, headache, vomiting and vertigo; (3) by excluding lesions of the motor, speech, visual and auditory areas, and their association tracts; (4) by special pressure and invasion symptoms—invasion by lesions growing from adjacent areas to those under consideration.

Lesions of the pre-frontal lobe, although this is one of the so-called latent districts of the brain, have in a large percentage of the carefully studied cases shown distinctive manifestations. The symptoms are largely psychical, and unfortunately the physician is not usually well trained to study such phenomena. Mental disturbances of a peculiar character occur, such as mental slowness and uncertainty, want of attention and control, and impairment of judgment and reason; closely studied, the inhibitory influence of the brain both upon psychical and physical action is found to be diminished. Memory is not seriously affected although a continuous train of thought cannot well be followed, and complex intellectual processes cannot be thoroughly performed. The results of experiments upon lower animals have not been very helpful towards determining the

existence of pre-frontal lesions, because psychical phenomena cannot be studied with accuracy in animals below man. Ferrier[1] however found after removal of the pre-frontal lobe a decided alteration in the behaviour of animals, difficult precisely to describe. They had apparently lost the faculty of intelligent observation. Horsley and Schäfer,[2] Hitzig,[3] and Goltz[4] have also observed apparent mental changes. Phenomena such as these do not measure in usefulness for the average diagnostician with such positive objective manifestations as hemianopsia or Jacksonian spasm, but they should be valuable aids in the hands of close observers. Among pressure and invasion symptoms motor aphasia, nystagmus, contra-lateral paresis and unilateral convulsions often occur late, particularly in cases of tumour and abscess. Given these symptoms, if now lesions of motor and visual regions can be excluded, it remains only to differentiate the lesions of the cerebellum and possibly some lesions of the temporal lobe.

In cerebellar cases, as shown by the studies of Nothnagel,[5] Seguin,[6] and others, the most positive symptoms are usually choked discs, optic neuritis or secondary atrophy, occipital headache, sometimes increased by percussion or pressure on the occiput or neck, vomiting, often apparently causeless in character; typical cerebellar titubation or other disorders of motion or gait; nystagmus and conjugate deviation; and among pressure symptoms one-sided paresis, occasional anæsthesia, sometimes disturbance of temperature, pulse, respiration, and deglutition.

Some lesions of the cerebellar hemispheres as in cases reported by Hun[7] give no localising symptoms.

It might be of value to attempt a contrast of the usual symptoms produced by lesions in these localities. In pre-frontal disease psychic symptoms of the peculiar character just described are usually present; in cerebellar affections they are as commonly absent, although irritative lesions of any part of the encephalon may, of course, cause emotional mental disturbance. The station or gait is not affected in pre-frontal cases: in cerebellar lesions, either typical titubation or some form of ataxia or staggering is usually present. Cerebellar titubation is characteristic of a destructive lesion of the middle lobe of the cerebellum, and when

[1] 'Functions of the Brain.' [2] *Op. Cit.* [3] Quoted by Ferrier.
[4] Quoted by Ferrier.
[5] *Topische Dagnostik der Gehirnkrankheiten*, p. 78. Berlin, 1879.
[6] *Jour. Nerv. & Ment. Dis.*, vol. xiv. No. 4, April, 1887, 217-235.
[7] *Albany Medical Annals*, May, 1888.

lesions of the cerebellar hemispheres are present, this results from pressure or encroachment. Bulbar symptoms and symptoms of pressure on the bulbo-spinal tracts, both sensory and motor, are somewhat frequently present in cerebellar lesions, particularly late in the history of a lesion of large size. With pre-frontal lesions of large size, the pressure and invasion symptoms are more likely to be disturbances of speech or smell, facial, brachial or aural paresis, and unilateral spasm. Carefully recorded cases show that nystagmus and conjugate deviation may be present in both pre-frontal and cerebellar cases; but in the pre-frontal cases the latter is more likely to be towards the side of the lesion presumably destructive rather than irritative; in cerebellar cases more probably the reverse. Headache is more likely to be frontal in the pre-frontal, and occipital in the cerebellar. Elevation of local surface temperature and tenderness on percussion have some but no great value in differentiation.

Auditory Localisation.

While auditory localisation remains in a somewhat uncertain state, on the whole, the evidence is in favour of the localisation of the cerebral centres or area, of hearing in the temporal lobe, and probably in its upper portion, that is, in the first or second, or in both the first and second, temporal gyres. The most recent and interesting discussion of this subject has arisen from the reports of the results obtained by Schäfer in his experiments on special sense localisation in the cerebral cortex of the monkey. Dr. Ferrier,[1] in reviewing the paper of Prof. Schafer, attacks the results obtained and the conclusions drawn by the latter, and contends stoutly for the localisation of the centre of hearing in the temporal lobe, and in accordance with his original view, more especially in the superior convolution of that lobe. Dr. Starr, in his consideration of aphasia, will doubtless discuss at length the question of auditory centres, naming centres, speech association tracts and speech centres, and I will therefore only briefly and for the purpose of completeness refer to this portion of the subject. Schäfer claims to have more or less completely destroyed the superior temporal gyre on both sides in six monkeys, and in one of the animals to have removed every trace of the convolution on both sides. In these cases he reports that hearing was not permanently affected, so far as it is possible to determine in monkeys, and concludes that the auditory faculty is not localised

[1] *Brain*, January, 1888.

in the superior temporal convolution. In one experiment of Horsley and Schäfer, the superior temporal convolution was almost completely removed on both sides; in this case the animal appeared to hear quite distinctly, so far as it went the experiment being at variance with the results of Ferrier and those of Munk. The experimenters however say that they do not claim to have obtained direct corroboration or refutation of the views of Ferrier, but regard the question as still open.

Ferrier reaffirms his position, and reproduces with some additional facts, his original experimental evidence in favour of auditory localisation in the temporal lobe, and particularly in its superior convolution. Munk, who is quoted both by Ferrier and Schäfer, extended the area for perception of auditory impressions over the entire temporal lobe; and Luciani believes that lesions of the temporal lobe in dogs produce abolition or impairment of hearing.

Gowers[1] refers to an interesting case of extensive tumour in which the oldest part was beneath the first temporal convolution. In this case convulsions, commencing by an auditory aura referred to the opposite ear, were a very early symptom. In another case of tumour of the first temporal convolution and Sylvian fossa, unilateral convulsions were preceded by a loud noise as of machinery. He notes that the loss of deafness in such cases is not permanent; that perfect compensation seems to be possible, presumably by the corresponding centre of the opposite side; and that each auditory nerve must be structurally connected with both hemispheres, although only the connection with the opposite hemisphere is habitually in functional action.

The pathological evidence with reference to the existence of auditory centres is not extensive or very decisive. That complete deafness from cerebral disease may occur probably requires, as Ferrier states, the existence of symmetrical lesions in both temporal lobes. It has however been clearly established by reported cases, that the so-called word-deafness may result from lesion of the upper convolution or two upper convolutions of the *left* temporal lobe. Cases in which the power to understand spoken words has been lost or impaired with lesions, in part at least, in the temporal lobe have been reported by various observers as, for instance, by Seppilli, Monakow, and Amidon.

Ferrier[2] refers particularly to two cases in support of his position, one reported by Shaw[3] and one by Wernicke and Fried-

[1] 'Manual of Diseases of the Nervous System,' p. 454.
[2] *Brain*, xii., London, April, 1888, p. 18.
[3] 'Archives of Medicine,' Feb. 1882.—Abstract in *Brain*, vol. v., 1882-3., p. 340.

lander.[1] Shaw's case, a woman, aged thirty-four, two months before her admission into his asylum, lost power in the right arm, and soon after had a sudden apoplectic seizure resulting in loss of speech and deafness. The loss of power in the right arm soon passed off. She became incoherent, more or less maniacal and demented. On admission she was found to be perfectly deaf and blind. She died of pneumonia a year afterwards. *Post-mortem* examination showed complete atrophy of the angular gyri and superior temporo-sphenoidal convolutions of both hemispheres. The gray matter of the atrophied regions had entirely disappeared, leaving the outer layer attached to the pia mater, with a cavity underneath formed at the expense of the gray matter. The cranial nerves were normal in appearance, the optic nerves showed increase of the connective tissue septa, atrophy of the nerve fibres, and spaces filled with a colloid-like material.

Wernicke and Friedlander's case, a woman, aged forty-three, had never suffered from deafness or affection of vision, and was attacked June 22nd, 1880, with right hemiplegia and aphasia. She remained in the hospital until August 4th, when she was discharged. At this time the patient could speak but unintelligibly and was sometimes believed to be intoxicated. She not only could not make herself be understood, but she could not understand what was said to her. She was received into the hospital again on September 10th, with slight paresis of the left arm. The right hemiplegia had entirely disappeared. The patient was looked upon as insane, and was absolutely deaf, so that she could not be communicated with. She died of an attack of hæmatemesis on October 21st. An extensive lesion was found in each temporal lobe, invading the superior temporal convolution on both sides. The rest of the brain and the cranial nerves exhibited no abnormality. It was proved that the patient had previously enjoyed excellent hearing. Her total deafness occurred rapidly in connection with the other indications of cerebral disease.[2]

[1] *Fortschritte der Medicine*, band I., No. 6. March 15, 1883.

[2] With Dr. Roland G. Curtin, of Philadelphia, I exhibited at the Philadelphia Pathological Society, the brain of a man for many years a deaf mute. A description of this brain, with illustrations, will be published with the reports of some other specimens referred to in this paper, in a forthcoming number of the *University Magazine*. On the whole, the gross appearances could be regarded as favouring strongly Ferrier's auditory localisation. The first temporal convolution of the left hemisphere was narrow and lacking in gyral elaboration; it was apparently distinctly atrophied or arrested in development. The first temporal convolution of the right hemisphere was smaller than usual, but it did not present the marked smoothness and diminution in size shown by the corresponding convolution of the other side. The brain was compared directly

Large Lesion of the Temporal Lobe.

With a large lesion of one temporal lobe, as tumour, hæmorrhage or abscess, the diagnosis is best made by a careful consideration of pressure and invasion symptoms in addition to those which are strictly localising in character. A single case recently published by Dr. Bodamer and myself will perhaps best illustrate this mode of diagnosis. The chief symptoms were severe headache, more localised in the temporo-frontal region; pain on localised percussion; impairment of sight and hearing; choked discs; dilatation of the right pupil, and three days before death paralysis of the left arm and paresis of the left leg, and aphasia. The autopsy showed a large lesion centred in the middle of the right second temporal gyre; it was a vascular glioma, having beneath and partly around it a cavity containing detritus and a large and evidently recent clot. It occupied a large portion of the interior of the right temporal lobe, but was strictly limited to it.

Macewen reports an operation on a somewhat similar case, of a lesion definitely localised in the temporo-sphenoidal lobe. A patient exhibiting symptoms of cerebral abscess had on the left side ptosis, stabile mydriasis, paresis of all the ocular muscles, with the exception of the external rectus, without external squint; on the right side, paralysis of the facial muscles, which retained emotional expressions to a slight degree, and power to close the right eyelid by an effort of will, although it remained partially opened during sleep. He had also paresis of the right arm, which during the few hours he was under observation before operation, had amounted to distinct paralysis. The leg remained normal, and there was no diminution of cutaneous sensibility.[1]

with half a dozen other specimens, normal and abnormal, and was examined by several brain anatomists and morphologists, who agreed with me as to the striking appearance of lack of development of the first left temporal. Other peculiarities of the temporal, third frontal, and central convolutions, the island of Reil, &c., were noted, and will be given in the description of the specimen.

[1] While this paper is passing through the press, through the courtesy of Dr. H. C. Wood, of Philadelphia, I have had the opportunity of seeing a case of large lesion of the right temporal lobe which shows how active symptoms of such lesions may be absent for a long period, or if present how they may differ from the pressure and invasion symptoms in the two cases above given. Dr. Wood will publish a full account of this interesting and important case. The patient had had for many months general symptoms of brain tumour, such as headache, choked discs, vertigo, &c., but no paralysis; and no loss of hearing that could be detected by ordinary tests. During the absence of the patient from the city, left homonymous hemianopsia developed, with pronounced contraction of the other visual fields. Wernicke's hemiopic pupillary inaction was carefully tested for, but was certainly not present. Operation was performed by Dr. D. Hayes Agnew for Dr. Wood, and a cyst in the right

Tumour of the Facial and Auditory Nerves.

At the meeting of the Neurological Society, of London, March 15, 1888,[1] Dr. Sharkey read notes and showed the brain of a case of tumour of the auditory nerve. During the discussion of this specimen, Mr. Horsley stated that the tumour might have been removed by an operation he had already advocated—namely, incision of the tentorium and ligature of the lateral sinus. I have made two dissections with a view of determining whether a tumour in the intra-cranial course of the facial and auditory nerves can be removed, and have concluded that it could, and best, not by operating above the tentorium as I understand Mr. Horsley's suggestion, but by operating below the tentorium, and then pushing aside or excising an outer segment of the cerebellar hemisphere.

Olfactory Localisation.

The location of the cortical centre of smell is still uncertain, but the pointings are all towards the region of the uncinate convolution and its immediate vicinity. Zuckerkandl, of Gratz, in 1887, published a monograph on the olfactory centre, the work being anatomical and physiological, not clinical. The anatomical portion is largely comparative, considering the brains of twelve varieties of animals besides man. One chapter describes the cornu ammonis region, another the olfactory lobes of animals and man, &c. The writings of Burdach, Treverinus, Huguenin and Ferrier are largely quoted. Zuckerkandl believes, but I think without reason, that the entire limbic lobe is the seat of olfactory sensation. Among other cases, he has collected two in which infants born without the olfactory lobe on one or both sides, showed arrested development of the horn of Ammon, and in both of which the gyrus fornicatus and hippocampus were small.

occipital lobe was discovered and removed. After death a large fibro-glioma was found occupying almost the entire second and third temporal convolutions, and also invading the central portion of the first temporal, the superior aspect of which was somewhat flattened by pressure. The fourth and fifth temporal convolutions were apparently not greatly encroached upon, the uncinate convolution escaping entirely. The patient had no loss of smell. The occipital cyst was evidently a radiation or a secondary result of the growth, due to softening from obliteration of blood vessels. This case emphasises the great importance of carefully studying pressure and invasion symptoms with reference to such comparatively latent regions as the temporal (particularly the right temporal) and pre-frontal lobes. The pressure in this case was evidently not exercised in the same direction as in the two cases referred to above. In a lesion of a so-called latent region, the pressure and invasion symptoms will depend both upon the nature of the growth, and on the particular direction in which it happens to develop.

[1] *Lancet*, London, April 7, 1888.

Zuckerkandl, in this view, is simply following Broca, who divided all animals into osmatics and anosmatics, or good smellers and bad smellers, and believed that the whole of the falciform or limbic lobe was the cerebral organ of smell. I cannot here go into the anatomical and physiological arguments bearing upon this subject. To one familiar with brain anatomy a knowledge of the relatively large size of the gyrus fornicatus and hippocampal region in an animal like man, in whom the sense of smell plays so comparatively an unimportant part, is an argument of some weight against the views of Broca. In osmatics however the hippocampal lobule or region of the amygdala—the uncinate convolution so-called—is very large, while in the anosmatics it is comparatively small. Ferrier's view is therefore probably correct that the only relationship which undoubtedly exists is between the olfactory bulb and the anterior portion of the hippocampal convolution. The anterior commissure has an anterior and posterior division, which connect respectively the olfactory bulb and the region of the hippocampal lobule and nucleus amygdala, a fact which also tends to prove that the anterior portion of the hippocampal convolution is the cortical organ of smell.

Electrical irritation of the hippocampal lobule or uncinate gyrus in monkeys, cats, dogs, and rabbits furnished Ferrier with significant indications of subjective olfactory sensations. This reaction, the same in all, as described by Ferrier, was a peculiar torsion of the lip and nostril on the same side. The experiment in which Ferrier produced destructive lesions of the hippocampal lobule, were, on the whole, unsatisfactory, but to a certain extent supported the view that this region contained the centre for smell.

Few autopsies have been recorded in which loss or diminution of smell has been present as the result of cerebral lesion. Ferrier, however, refers to cases reported by W. Ogle, Fletcher and Ransome, of the occurrence of the loss of smell in the left nostril with right hemiplegia and aphasia; and he also alludes to cases reported by Hughlings Jackson. From a study of these cases he believes that we have reason to regard anosmia as probably due to softening of the region of the hippocampa lobule. The connections of the olfactory tract are with the hemisphere on the same side. In some cases of cerebral hemianæsthesia, as in the hysterical and alcoholic forms, impairment or loss of smell is present and seems to be contra-lateral. This, Ferrier believes, may be explained on the supposition that the defect was due to an anæsthetic condition of the nostril, either

from lesion of the fifth nerve or of the centre for cutaneous sensation in the cerebral hemisphere. Close testing in such cases would probably show that the olfactory sense was not absolutely abolished.

Various cases have been reported with autopsies in which the presence of an olfactory aura has been accompanied by a lesion of the temporal lobe, and these cases, on the whole, point to the lower convolutions of this lobe as the probable seat of the centre of smell.

Hughlings Jackson has written various papers showing the great importance of studying all forms of aura in order to be able better to localise the lesions in the encephalon. As early as 1876, and again in 1879, he considered the varieties of epilepsy in which not only crude sensations or warnings of smell, taste, &c., were present, but also a more elaborate mental condition which he speaks of as the "dreamy state." As theoretical as such discussions may seem at first sight, he clearly shows that they are of practical value not only in leading to an early recognition of epilepsy, but also in localising lesions in either epileptic or epileptiform cases.

I have had several epileptic patients in whom the attacks were initiated by an odour usually offensive. Jackson [1] gives interesting histories of three such cases in which attacks were ushered in by a crude sensation warning of smell, accompanied sometimes by other warnings, as epigastric sensations and the dreamy state. These cases are of great clinical interest but are not accompanied by autopsies. He refers, however, to the necropsy of a woman who had had paroxysms with the dreamy state and crude sensation warnings of smell. She had left hemiplegia and double optic neuritis. The autopsy showed a tumour in the right tempero-sphenoidal lobe.

Allan McLane Hamilton [2] has reported the following case of softening of the temporo-sphenoidal lobes, in a woman of forty years. The patient suffered from the age of ten from epileptic attacks, which occurred four or five times a year, and consisted of general convulsions. The first attack occurred after a fall when she struck her head, and was unconscious thereafter for some hours. No scar was visible on the head. She always had an aura of a peculiar character before the attack. She suddenly perceived a disagreeable odour, sometimes of smoke, sometimes of a fœtid character, and quite uncomplicated by other sensory warnings. She died of phthisis.

[1] *Brain*, July, 1888.
[2] *Am. Jour. Med. Sci.*, April, 1884. Quoted by Starr.

On *post-mortem* examination the dura mater was thickened and opaque in spots, and at the base was adherent to the temporo-sphenoidal lobes. The adhesion of the membranes was most marked on the right temporo-sphenoidal lobe somewhat posterior to its apex. At this point a decided shrinkage of tissue was discovered with depression, the duration involving the uncinate gyrus and parts of the adjacent convolutions. The basal ganglia and motor tracts were normal, and the olfactory nerves were not involved.

Worcester[1] reports the case of a farmer, aged thirty, who had had epilepsy for two years before admission to the hospital. The case presented no special features until January 26, 1878, when after a severe convulsion the man remained in a state of alarming collapse. Radial pulse was almost imperceptible, surface cold, and there appeared danger of immediate dissolution. He rallied somewhat under stimulants, but remained for three days in a stupid condition unable to be long out of bed. Shortly after the attack slight innervation of the right side of the face was observed, but only when the muscles were called into action as in talking and smiling. On February 11th he regained his ordinary mental condition. No paralysis was discovered except as above mentioned, and no impairment of sensibility except a transient numbness of the hand at times. For several days hallucinations of smell—at first constant, afterwards transitory—were present. Once he imagined the room was full of smoke. He fancied at times there was an odour like the vapour of alcohol passing quickly. He thought this took the place of a convulsion. No test was made of his sense of smell. No marked changes occurred until his death on February 28th, after a series of tonic convulsions, with marked opisthotonos, affecting chiefly the muscles of the back.

The autopsy revealed, on inspection of the inferior surface of the brain, a small red spot of softening at the most prominent point of the left gyrus uncinatus. The brain was not opened until it had been hardened in alcohol. A focus of softening existed in the white matter of the anterior part of the left temporal lobe, extending to the surface, externally, and internally involving the pes hippocampi in the floor of the descending cornu of the lateral ventricle. The portion of the hippocampus major not discoloured was swollen and softened. A very small focus of softening, without discolouration, about the size of a large pea,

[1] *American Journal of Insanity*, for July, 1887.

was found in the white matter of the frontal lobe on the same side. No other gross lesions were discovered, but the perivascular spaces were very generally dilated, so as to give thin sections of the brain a worm-eaten appearance.

Gustatory Localisation.

Our knowledge of a cerebral centre for taste is even more unsatisfactory and undecided than that for an olfactory centre. Morphology, anatomy, physiology and pathology combine to indicate that this centre is probably situated close to and in the same lobe of the brain as that for smell. The experiments of Ferrier seemed to show that affections of both taste and smell were evidently connected with lesions of the hippocampal lobule and its neighbourhood. "It was noted in connection with electrical irritation of the lower extremity of the temporo-sphenoidal convolutions in the monkey, and of the same region in the brain of a cat, that the movements of the lips, tongue, cheek-pouches and jaws were occasionally induced—phenomena which might be regarded as indications of the excitation of gustatory sensation. This interpretation receives support from the above described results of destructive lesions; and we have, therefore, reasonable grounds for concluding that the gustatory centres are situated at the lower extremity of the temporo-sphenoidal lobes, in close connection with those of smell. This would enable us to explain the occasional occurrence in man of anosmia and ageusia as the result of severe blows on the head, especially the vertex. A blow in this region causes counter-stroke of the base of the brain, particularly in the region of the olfactory centres."

Dr. James Anderson[1] has recorded a case of epilepsy in which from symptoms, ocular and cerebral, detailed in his report, he correctly predicated tumour and its position. The patient's dreamy state was associated with a rough, bitter sensation in his mouth. It is the only case published, according to Jackson, in which a necropsy has been had revealing any local morbid changes in a case of the variety of epilepsy mentioned. Dr. Anderson refers to a case, closely like that of his own patient, recorded by Mr. Nettleship. In the report of this case however the dreamy state was not mentioned; there was a crude sensation warning in the patient's fits—a sudden feeling of suffocation in the nose and mouth.

As our special subject is the practical relations of cerebral

[1] *Brain*, October, 1886. Quoted by Hughlings Jackson in *Brain*, July, 1888.

localisation, I cannot forbear to recall here the advice of Hughlings Jackson[1] on the great practical importance of the close study of epileptic seizures. These remarks are made in connection with the discussion of the different varieties of aura—crude warnings of smell and taste, intellectual aura of the dreamy state, temporary word-blindness or word-deafness, noises, flashes of light, hallucinations, &c. No better neurological work, Jackson holds, can be done than the precise investigations of epileptic paroxysms. The efforts should be to describe *all* that occurs in the paroxysms. Epilepsies are as numerous as are paroxysms beginning with different warnings. The warning is the first event from or during the onset of the epileptic discharge; it is the clue to the seat of the discharging lesion. Using the term suggested by Seguin, I might interpolate here that it is the sensory "signal symptom." The discharging lesions have as many different seats as there are different warnings of the paroxysms.

"Before we can make good generalisations," says Jackson, "we must carefully analyse. To group together as 'visual warnings' colour projections, apparent alterations in the distance of external objects and 'dreamy states' with definite scenes, is generalising without previous analysis, and is an attempt to organise confusion; they are exceedingly different things. He who is faithfully analysing many different cases of epilepsy is doing far more than studying epilepsy. The highest centres ('organ of mind'), those concerned in such fits, represent all, literally all parts of the body sensorily and motorily, in most complex ways, in most intricate complications, &c. A careful study of many varieties of epileptic fits is one way of analysing this kind of representation by the 'organ of mind.' Again, it is not, I think, an extravagant supposition that there are, after slight epileptic fits of different kinds, many temporary morbid affections resembling those persistent ones produced by destructive lesions of different parts of the cortex. To illustrate for a moment by epileptiform seizures; there is temporary aphasia after some fits beginning in the face or hand (more 'elaborate' utterances, I think, when the exact starting point is in the ulnar fingers); this is the analogue of aphasia from a destructive lesion (softening, &c.). To return to epilepsy. There is, I am convinced, in or after certain paroxysms of epilepsy temporary 'word-blindness;' certainly in one patient of mine who had a 'warning' by noise. I could not make out

[1] *Brain*, July, 1888.

that this patient was at the same time 'word-deaf,' but thought his temporary deafness was ordinary deafness. Still there may have been word-deafness. In another patient who called his attacks 'losses of understanding,' there was clearly both 'word-deafness' and 'word-blindness,' with retention of ordinary sight and hearing. This patient's attack used to begin with a warning noise, but he has recently had his 'losses of understanding' without that warning."

Jackson holds that there is some local disease in every epilepsy, some pathological process productive of high instability. His views on the arterio-cortical pathology of some varieties of epilepsy or epileptiform seizures are of great importance to those who are concerned not only in locating the site of a discharging lesion, but also in deciding whether such lesion shall be removed by operation. Sometimes, in the cases already operated on, even when the most careful and doubtlessly accurate localisation has been made no gross lesion has been discovered, and yet even in these cases a true gross lesion may have been present—if the plugging of arterioles can be regarded as a gross lesion. Jackson believes that most cases of epilepsy proper are due to the plugging of arterioles. His views upon this question are full of suggestive value. "Centres of taste and smell," he says, "lie, according to Ferrier's localisation, in the region of the posterior cerebral artery, whilst, still according to his localisation, the centres for hearing and part of the centre for sight (angular gyrus) lie in the region of the middle cerebral. Hence, if arterial plugging be the pathology, it may be that we have different varieties of epilepsy proper, according as arterioles are plugged in different vascular regions. The variety of epilepsy I am remarking upon in the text may be owing to morbid changes in the district of the posterior cerebral. But tumours would grow regardless of vascular regions. I suggest that cases of epilepsy with mixed warning (of smell or taste along with the warnings of colour) are more likely to be owing to tumour or other gross organic disease."

Handicapped by the embarrassing proportions of my subject, I have imperfectly presented it for consideration; but trust that my remarks may open lines of discussion to those here present far better fitted than I to enter the lists in such a debate. These practical discoveries in cerebral localisation, with the achievements of antiseptic surgery, constitute the grandest triumphs that adorn the history of our noble science and art of medicine.

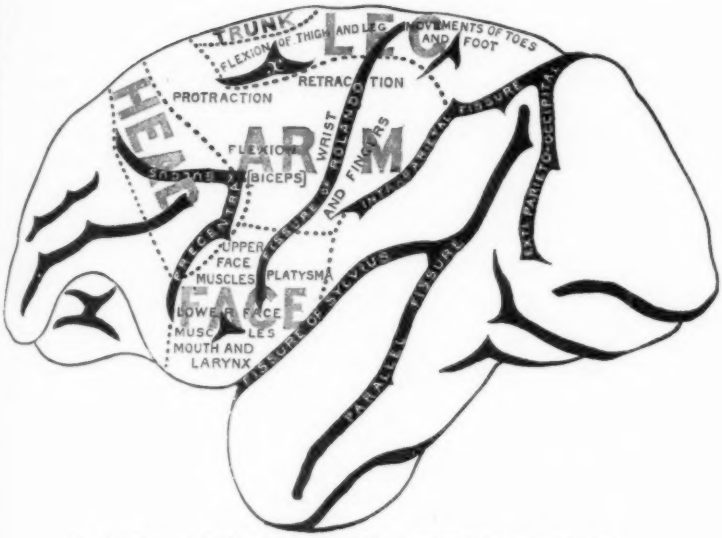

Fig. 1.—Lateral Surface of Brain of Monkey (Horsley and Schäfer).

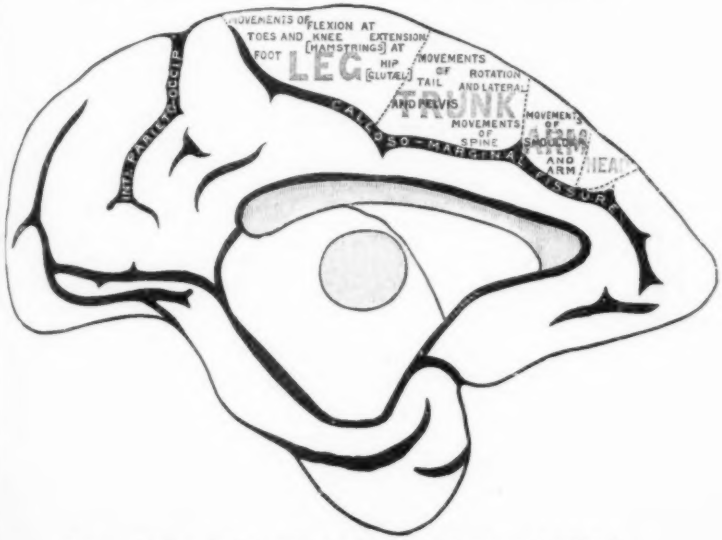

Fig. 2.—Median Surface of Brain of Monkey (Horsley and Schäfer).

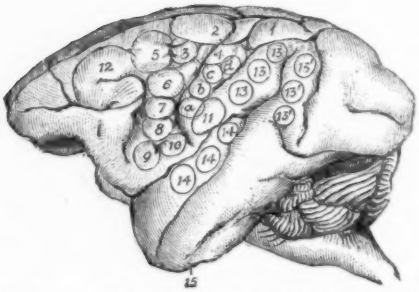

Fig. 3.—Lateral Surface of Brain of Monkey (Ferrier).

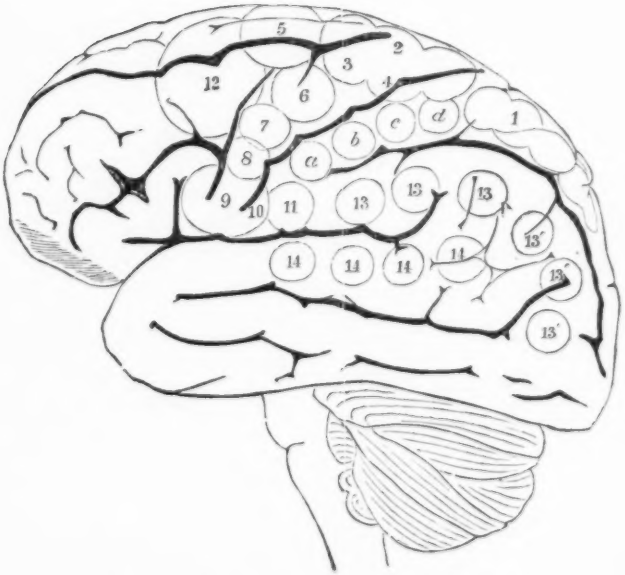

Fig. 4.—Lateral Surface of Human Brain (Ferrier).

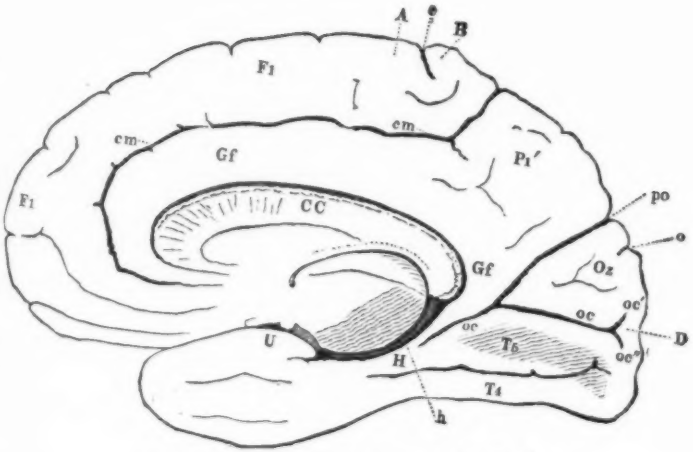

Fig. 5.—Median Surface of Human Brain (Ferrier).

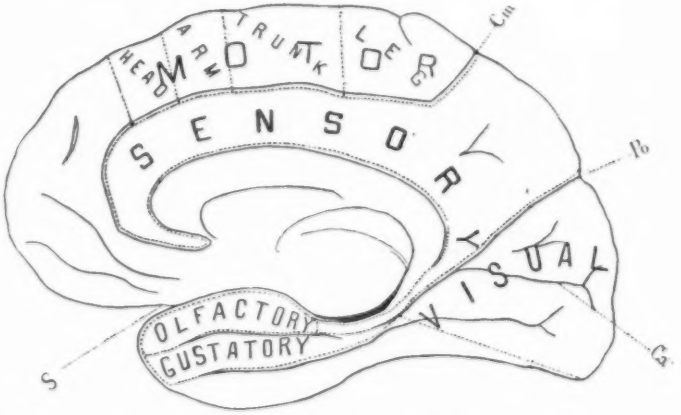

Fig. 7.—Areas of the Mesial Aspect of the Cerebrum.

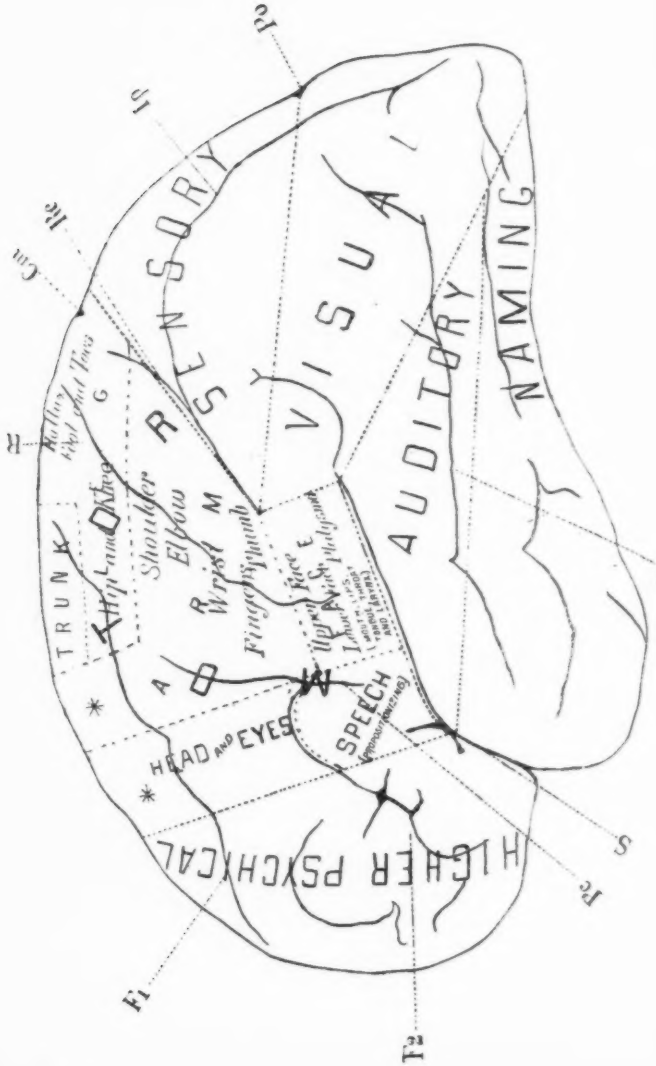

Fig. 6.—Areas of the Lateral Aspect of the Cerebrum and Sub-divisions of the Motor Area.

Fig. 6.—Areas of the Lateral Aspect of the Cerebrum, and Sub-divisions of the Motor Area.

Reviews and Notices of Books.

Les Agents provocateurs de l'Hystérie, By GEORGES GUINON, *Thèse, Paris*, 1889. 1 vol. in 8, 392 pages. Librarie du Progrès Medical, and Lecrosuier et Babé, editeurs, Paris.

THIS work, done under the direction of Professor Charcot, is consecrated to the study of the causes that preside over the growth of hysteria, and also to the different modes of development of neurosis under their influence.

The first part, exclusively clinical, contains the enumeration of the different *exciting causes* ("agents provocateur"), with numerous observations in their support. As to what concerns each of them the author has attempted often with success to find in the old authors some examples of hysteria developed under the influence of exciting causes, the action of which was unknown at the time of the outbreak. In other cases on the contrary hysteria was denied and the nervous disturbances that characterise it were regarded as proceeding from some exciting disease of which they constituted, according to the authors, only a symptom. M. Georges Guinon shows us that hysteria, produced by a certain number of different causes, has always existed, and that taken altogether it is through mistakes in doctrine or in interpretation that it has never been judged at its right value.

Let us only enumerate here the different exciting causes that the author has studied, the lack of space preventing us from following him in the particular study he makes of each of them. Amongst them some have been known for a long time; others—and it is a very interesting point—have had their action revealed quite recently through the researches of Professor Charcot. These exciting causes are: *moral* emotions, attempts at hypnotism, nervous shock, earthquake, traumatism, shock of lightning; general and infectious diseases, such as typhus fever, pneumonia, scarlet fever, rhuematic fever, diabetes, paludal infection, syphilis;

morbid states characterised by a considerable weakness of the patient, such as hæmorrhages, physical or intellectual over-exertion, onanism or venereal excess, aneury and chlorosis; intoxications (particularly that by the chloroform in surgical anæsthesia) or chronicals (lead, mercury, sulphuret of carbon) or acute diseases of the genital apparatus; several diseases of the nervous system (sclerosis in disseminated patches, tabes dorsalis, Friedreich's disease, progressive pruritive myopathy, and low compression of the marrow by Lott's disease). This list, already long, is as the author shows, far from being at an end, for the simple reason that the exciting causes are but occasional, insignificant, and not at all specific.

It is partly to the demonstrations of this proposition that the second part of the work of M. Georges Guinon is devoted. He establishes first of all that the disease of which he has given so many examples in the first part is in reality hysteria. But he remarks also, in answering criticism made abroad, and particularly in Germany, that if the exciting causes develop hysteria, they can also give life to other nervous disorders, neurasthenia particularly, whose combination with hysteria constitutes some really complex cases that have been and are still falsely interpreted and considered as special cases in the nosography, under the names of traumatic, taxic neurosis, &c. This combination of different affections under the influence of the same causes is easily understood if one takes in consideration that the existing agents, as the author shows us, are but common occasional causes whose influence can be found frequently in general pathology. But it must be noticed that exciting causes can sometimes give a certain impression to the accidents belonging to the neurose. An hysterical man acted on by lead, for an example, will have an hysterical monoplegia presenting at first sight all the characteristics of the real paralysis by lead, but having also those of hysteria, upon which in reality it directly depends.

M. Georges Guinon then studies the different modes of development of the neurose under the influence of exciting causes, and shews us, with examples, that it may appear several years after their action. Finally he tries to define the mode of action of the exciting causes. In certain cases the outbreaks are caused by phenomena of *auto-suggestion* occurring to the patient against his will. In other cases that are not to be justified by this explanation, the author admits the hypothesis of something

wrong in the general nutrition, and particularly the nutrition of the nervous system presiding over the outbreak of the symptoms of neurosis.

Among the eighty-five observations that form here the most solid of foundations, a great number are hitherto unpublished, and are drawn from the author's personal experience. Taken with the greatest care, and accompanied by drawings showing the state of the cutaneous sensibility and of the eyesight, they form a collection of much more than ordinary value. Moreover, this work is based on extensive bibliographic researches, which are utilised at the end of the volume to form an index that is without doubt the richest that has hitherto been published on the subject. The foreign publications occupy, as we have observed, an important space, so that for all that has any relation to such important questions as taxic hysteria, railway accidents, paralysis dependent on idea, &c., &c., one is sure to find there references to a great number of works which otherwise would have been exceedingly difficult to discover.

Such is, in its principal lines, the arrangement of M. Georges Guinon's work. Summary as this exposition may be, it is sufficient to show that this work is one of the most important of those that have been suggested by Prof. Charcot in recent years. It does honour equally to its author and to the *École de la Salpêtrière*. After the period of sure and delicate analysis applied by the illustrious teacher to the study of hysteria, this work seems to come as an introduction to a new era—an era of synthesis through which we shall learn to know, not only the various manifestations of neurosis, but its very essence.

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Abstracts of British and Foreign Journals.

Current Nerve Anatomy and Physiology.—Titles and Indication of Contents of recent Papers. By Alex. Hill, M.D., Master of Downing College, Cambridge.

Anatomy Schema.

1. The Structure of the Mammalian Brain. Jelgersma (*Morphologisches Jahrbuch* xv. 1, pp. 61-85, Part iv., June, 1889).

Submits a scheme of the plan of formation of the mammalian brain, based on comparative anatomy. After leaving the indifferent brain of the reptile we find as we ascend two diverging lines of development: 1. The great growth of the "basal ganglia" in birds with rudimentary cortex cerebri. 2. The great growth of cortex in mammals. Author shows that certain changes in medulla are associated with the growth of the cortex and groups these parts together as intellectual tracts. These tracts include the cortex of cerebrum and cerebellum, the pons, nucleus olivaris &c. He considers that the growth of the convolutions and sulci is altogether independent of external influences, and due simply to the necessity for finding greater accommodation for the grey matter and white tracts.

Anatomy.

2. Collected Posthumous Works of v. Gudden (published *Grashey, Wiesbaden*, 1889).

Brain: Topography.

3. Brain Growth, Cunningham (*British Medical Journal*, No. 1,465, p. 190, 1889, *Royal Academy of Medicine in Ireland*).

Notice of the exhibition of several more specimens illustrating the growth of the brain in children.

4. Experiments in Cranio-cerebral Topography. Anderson and Makings (*J. of Anat. and Phys.*, April, 1889, pp. 455-466).

Brain: Geology.

5. Asserted Petrifications of the Brain, Schiff (*Arch. d. sciences phys. et nat.* xx. 12, p. 582).

Weight.

6. Brain Weight of New-born Children. Mies (*Wiener klin. Wochenschr*, 1889, 2, p. 39).

Evolution.

7. On the Relation between the Structure, Function, Distribution and Origin of the Cranial Nerves; together with a Theory of the Origin of the Nervous System of Vertebrata. Gaskell (*Journal of Physiology*, x. 3, pp. 153-211, pls. xvi.-xx, *April*, 1889).

Cf. Paper in this Journal xlv. and xlvi., July, 1889, in which Gaskell explains his views as to the origin of the vertebrate nervous system. In this paper Gaskell also gives an account of the histological character of the third and fourth nerves, showing that they contain amongst their fibres tissue not found in other nerves. This tissue appears vestigial, representing apparently degenerated "stationary" or root ganglia. Nerves III. and IV. are therefore complete segmental nerves, the sensory parts of which have degenerated and been replaced in their distribution by sensory elements of V. The ramus ophthalmicus profundus is not a separate dorsal root, but a part of V. which has come up from a hinder segment for the purpose of replacing lost sensory nerve roots. III. and IV. are the primitive segmental nerves of the first and second segments.

VII. also shows traces of a lost sensory ganglion. It therefore corresponds to a complete segmental nerve. The fibres from the back of the oculo-motor nucleus which pass down the posterior longitudinal bundle and out of the brain, with the roots of VII. to supply the m. orbicularis palpebrarum, constitute the splanchnic ganglionated efferent portion of the oculo-motor and trochlearis segments. In his earlier papers Gaskell regarded the large fibres of VII., which form a group by themselves (somatic efferent division of the nerve) as destined for mm. stapedius et levator palati. This he finds is an error. He is unable therefore to find the somatic efferent fibres of the hyoid segment.

The paper is illustrated with numerous reproductions of sections, drawings and diagrams in colour.

Comparative Anatomy.

8. The Brain of the Chimpanzee. Giacomini (*Atti della R. Academia delle Scienze di Torino*, xxiv. 24 pp. 4 *lithographs*, *June* 23, 1889).

Detailed account of surface of corpus callosum and base of brain.

9.—Internal Structure of the Amphibian Brain. Osborn (*Journal of Morphology*, vol. ii., No. 1, pp. 51-96, pls. iv.-vi. 1888).

Results of observations on Amphiuma, Cryptobranchus, Necturus, Siredon, Proteus, Rana and Siren. The following are some of the most important results arrived at:—1. The determination of the chief motor and sensory nuclei of the fifth and tenth pairs, which enables him in some degree to homologize the intra-axial elements of the vagus and trigeminal systems, and demonstrates the independence of the auditory system and system of the motor nerves of the eye-ball. 2. The discovery of a new tract and nuclei—the *fasciculus communis*—common to the tenth, ninth, and seventh (or eighth) pairs. 3. The determination of the relations of the posterior longitudinal fasciculus (uncrossed Müllerian fibres) to the eighth, sixth, fourth and third nerve tracts and of the nucleus of the latter nerve with the fibres of the posterior commissure. 4. The passage of a portion of the descending trigeminal tract through the cerebellum, and the direct connection of this bundle with the large mesencephalic nucleus. 5. In the encephalon the determination of the direct motor tract to the prosencephalon and of direct sensory tracts to the mesen- and dien- cephalon.

The brain forms are described in great detail and well illustrated. It is impossible to give an abstract of the anatomical work, but certain conclusions may be mentioned. Osborn suggests that the cerebellum is primitively intersegmental, and secondarily acquires a functional importance equivalent to that of the other segments. The early constriction of the brain roof, which gives rise to the four vesicles, is for the accommodation of three nerve-fibre tracts decussating dorsally, viz., the superior and posterior commissures and the cerebellum, which in their primitive condition have a serial homology. With regard to the intra-axial origin of cranial nerves in amphibia, Osborn comes to the following "provisional conclusions:" (1) That there is a close similarity between the disposition of the nuclei and tracts of the V.-VII. and IX.-X. groups—the nerves of these groups being complementary to each other, and together apparently containing fibres from two sensory nuclei and from two motor nuclei. The extreme dorsal and ventral nuclei are composed respectively of unmistakeable sensory and motor cells. (2) III., IV., and VI. pairs form a special system not homologous with (1). (3) VIII., as a nerve of special sense, has either no homology or an incomplete homology in the arrange-

ment of its sensory nuclei and tracts, with the sensory elements of the vagus and trigeminal systems. The fine fibres of the urodele cerebellum are in part decussating tracts of the auditory nerve, and the coarse fibres are non-decussating descending tracts of the trigeminal nerve. The cerebral commissures contain decussational fibres. There is a very close similarity between the amphibian (urodele) and dipnoan brain, both in external and internal structure.

COMPARATIVE ANATOMY: FISHES.

10. The Homologies of the Inferior Lobes of the Brain in Fishes. CHATIN (*Comptes Rendus*, cviii., 12, p. 628).

COMPARATIVE ANATOMY.

11. The Nervous System of the Decapodous Crustaceans and its Relation with the Circulatory Apparatus. BOUVIER (*Annales des Sci. Nat.* vii., 1 and 2, pp. 72-96, pl. 7, 1889).

COMPARATIVE ANATOMY.

12. Neurology of the Prosobranchiata. BROCK (*Zeitschr. f. w. Zoologie*, xviii., 1, pp. 67-84, pls. vi. and vii., 1889).

COMPARATIVE ANATOMY.

13. Ventral Nervous Mass of Fissurella. BOUTAN (*Archives de Zool. Exper.* vi. 3, pp. 375-433, pls. xxi., xxii., xxiii., 1888).

COMPARATIVE ANATOMY.

14. The Nervous System of Amphiptyches Urna. MONTICELLI (*Zool. Anzeiger*, 302, p. 142, 1889).

DEVELOPMENT.

15. Development of Amphibians, chiefly concerning the Central Nervous System, with additional Observations on the Hypophysis, Mouth, and the appendages and Skeleton of the Head. ORR (*Quart. Jour. Micros. Sci.* cxv., pp. 295-325, pls. xxvii.-xxix., *Dec.*, 1888).

Embryos of Amblyostoma, Triton, and Rana. Conclusions:—The central nervous system of amphibians first appears as a transverse epiblastic thickening dorsal to the mouth-fusion. This transverse thickening is continuous with elongated thickenings, which lie on either side the mid-dorsal line. The transverse thickening is the cause of the primary cranial flexure. It corresponds to the region at the base of the brain between the infundibulum and optic groove. The first nerve-fibres are formed on the inner side of

the longitudinal thickenings. Subsequently a continuous ventral commissure, anterior and posterior commissures are formed. The fibres of the optic nerve are intimately connected with the main bundle of fibres in the region of the primitive transverse thickening. The anterior band of nerve-fibres corresponds in situation with *Edinger's* "Commissur der basalen Vorderhirnbündel." Its large size indicates that it was one of primary importance. The commissure in the adult brain is probably a rudiment of the same with changed relations and functions.

COMPARATIVE ANATOMY CORTEX.

16. Convolutions and Sulci of Great Brain of the Dog. ELLENBERGER (*Archiv. f. wiss. u. prakt. Thierheilkunde*, XV., 3 & 4).

CORTEX: PHYSIOLOGY.

17. The Results of Section of the Association Fibres in the Dog's Brain. EXNER AND PANETH (*Pflüger's Archiv.* xliv. p. 544, 1888).

CORTEX: IRRITABILITY.

18. Excitability of the grey substance in the motor area of the cerebral cortex. ADUCCO (*Archiv. ital. de Biologie* xi. 1, p. 122, Jan., 1889.

CORTEX: LOCALISATION.

19. The Physiology of the Motor Area of the Cortex. BECHTEREW (*Russian Arch. of Psychiatry* ix. 2, 3, and x. 1, 2, 1889.)

CORTEX: MOTOR.

20. The Nervous Centres in a Man from whom a limb had been amputated. BIGNAMI AND GUARNIERI (*Boll. Della R. Accad. Medic. di Roma. Abs. in Archivio ital. per le Malat. Nervosi* xxvi. p. 40, 1889).

A man 45 years old, from whom left arm had been amputated eleven years before. Atrophy of posterior column and whole of grey matter in cervical region on the left side and of the right column *of Gower*. Atrophy of the cortex of both central convolutions on the right side.

CORTEX: EPILEPSY.

21. The Relation of the posterior part of the Cortex to the Epileptic Fit. UNVERRICHT (*Deutsch. Arch. f. Klin. Med. Bd.* 44, 1889).

There has been some controversy between U. and Rosenbach as to the meaning of the convulsions which may be provoked by

stimulating the occipital cortex. U. considers these fits as distinctly of cortical origin, although distinguished by one-sidedness. He thinks they are due to the propagation to the motor area of stimuli originating in the occipital cortex. These fits are liberated by stimulating the most posterior region in which such a stimulus is effective—the posterior second longitudinal convolution on the inner side of the vision sphere. Stimulation with short weak currents induces movements of the eye balls to the opposite side—later widening of the pupil and opening of lids. With longer action an epileptic fit is liberated. The fit begins with nystagmus and spreads in the order in which the current would extend supposing it to have passed from the occipital region to the frontal cortex in the first place and there to have originated the movements of the eye balls by cortical action in this spot in the front of the motor area : nystagmus, movements of ear, orbicularis, muscles of cheeks, tongue and extremities; bending of vertebral column, widening of pupils, and then extension of the fit to the opposite side of the body. Sometimes movements of posterior extremities precede the movements of orbicularis. If the orbicular centre is severed from the centre for the lateral movements of the eye by a sharp cut the course of the fit is altered, and contraction of the orbicularis ends the one-sided fit. The fit affects one side before it spreads across to the other.

Paper is illustrated with photograph showing alterations of fit produced by cutting away motor area (in dogs). If whole motor area is removed movements of orbicularis and ear of the opposite side follow the nystagmus—then a pause as long as would have been required for the spasm to travel down to the extremities, and after this the spasm passes across to opposite hind foot and gradually ascends body.

22. The Question of the Epileptogenous property of the Posterior Part of the Brain. Rosenbach (*Neurologisches Centralblatt*, 1889, *No.* 9, pp. 249-257).

Contra Unverricht (cf. supra). Rosenbach concludes that because epilepsy is no longer produced by stimulating the occipital cortex after removal of the motor area, it is due, when this area is intact, to spread of stimulus. U. believes that it has its proper origin in the spot of cortex stimulated. The paper is devoted to arguments against U.

Epilepsy.

23. Lessons on the Motor Functions of the Brain and on Cerebral Epilepsy. Francois-Franck (Paris, 1889).

Graphic analysis of simple movements produced by electrical

excitation of the cortex. Latent period, form, and duration of curves. Convulsions provoked in the same way and studied with similar apparatus. March of the epileptic seizure and its characters, whether complete, incomplete or abnormal. Study of the characters of the disorders of the circulation of the pupils and of secretions which occur on stimulating the cortex of a curarized animal, and conclusion that in these cases the seizures are epileptic, the organic system alone participating in the epilepsy. Effects of lesions of the brain upon motor functions. Theoretic considerations; proper excitability of cortex as peripheral impressionable field.

CORTEX: VISION.

24. Case of Cortical and Sub-cortical Disease of the Occipito-angular Region producing Hemianopia. SHARKEY (*Trans. Ophthalmo. Society*, viii., p. 304).

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25. The Relation of the Occipital Lobes of Newborn and Young Animals to Movements of the Eyes. DANILLO (*Vorläufige Mittheilungen Wratsch, No.* 48, 1888).
26. Influence of Cortical lesions on Vision (*concluded*). LANNEGRACE (*Archives de Médecine Expérimentale, &c.*, 1889, ii., p. 289).

Experiments on monkeys. 1. Lesion of one occipital lobe seems to have no effect on vision (agrees with *Ferrier, Yeo, and Munk*). 2. Lesion of one gyrus angularis affects vision of opposite side, but effect is transitory. 3. Lesion of the gyrus angularis and occipital lobe affects vision with both eyes, producing crossed amblyopia as well as homonymous hemiopia (agrees with *Ferrier*, and *Yeo*). 4. Lesion of the Rolandic convolutions disturbs vision. 5. Successive lesion of both angular gyri results in no loss of sight. 6. Destruction of both occipital lobes causes disturbances of vision but not blindness. 7. Destruction of both occipital lobes and one angular gyrus does not cause complete blindness (in accordance with *Ferrier*). 8. Destruction of both occipital lobes and both angular gyri does not necessarily produce complete loss of vision as *Ferrier* and *Yeo* assert. In one case L. obtained this result, in another case not.

L. concludes that to completely destroy vision in monkeys the injury must extend farther, into the motor zone. In chapter four L. gives a theory of hemiopia and amblyopia, and the rest of the paper is occupied with a discussion of the course of optic fibres.

CORTEX: HEARING.

27. The first temporal convolutions right and left in a person deaf with the left ear. MANOUVRIER (*Revue Philosoph.* xiv., 3, p. 330).

CORTEX: SALIVARY SECRETION.

28. Secretion of Saliva on stimulating the Cortex. ECKHARD (*Neurologisches Centralbl. Feb.* 1, 1889).

29. The question of the Cortex areas which induce secretion of saliva. BECHTEREW and MISLAWSKI (*Neurolog. Centralbl., April* 1, 1889).

CORTEX: TEMPERATURE.

30. Thermo-electric observations upon the Cerebral Cortex in various emotional states. TANZI (*Revista Speriment. di Freniatr.* xiv. p. 234, 1888).

Variations of 1°-2° and in some instances 3° C. observed in the dog, independently of alterations in pulse and respiration.

CORTEX: DEGENERATION.

31. Changes in the Cerebral Cortex in a case of blindness and deafness developed in early childhood. TOMASCHEWSKI (*Centralbl. f. Nervenheilkunde* xii. v. 1, p. 21, 1889).

CORTEX: ELECTRIC CONDUCTIVITY.

32. Induced unipolar currents in studying the electrical excitability of the brain. NEGRO (*Arch. ital de Biol.* xi. ii. pp. 212-225, *March*, 1889).

CORPUS CALLOSUM.

33. A case of absence of Corpus Callosum in the human brain. BRUCE (*Reports from the laboratories of the Royal Coll. of Physicians, Edinbro'*, 1889. *Medical Record*, 1889, *No.* 15, p. 87).

PINEAL BODY.

34. Multiple pineal eyes of the blind-worm. DUVAL and KALT (*C. R. Soc. de Biologie* 1889, p. 85, *Feb.* 9).

HYPOPHYSIS.

35. Certain stages in the development of the Hypophysis Cerebri. LECHE (*Verh. d. Biologie in Stockholm I.* 3, *Dec.*, 1888).

CORPUS MAMMILLARE.

36. Anatomical Remarks on a Case of Atrophy of the left Corpus Mammillare. WINKLER and TIMMER (*Nederl. Tijdschr. voor Geneesk.* 26, 1888).

Left hemisphere atrophied in its convolutions, but not much so on the base. Left corpus mammillare atrophied. Fimbria in front of descending horn well developed, but very thin where it began to curve. Pulvinar and anterior tubercle of thalamus were atrophied, and the thalamus shorter than normal. No distinct atrophy of the optic nerve or its connections. The cortex of the gyrus hippocampi and subiculum cornu Ammonis were slightly atrophied, the left crus fornicis and columna ascendens were strongly so. Observations upon the course of the columna ascendens of the right fornix.

PYRAMIDAL TRACTS.

37. The Pyramidal Tracts in certain Mammals. LENHOSSÉK (*Anat. Anzeiger, April* 20, 1889, pp. 208-219; *wood-cuts*).

Mouse, guinea pig, rabbit, cat; the pyramidal tracts much less developed than in man, as estimated by relative cross-sections at birth. They also occupy different situations in different animals. In the mouse and rat they lie in the posterior columns, in the rabbit and cat in the lateral columns, in man partly also in the anterior columns. In all animals observed the tracts completely cross.

38. The Intercrossing of the conducting paths serving voluntary movements. BROWN-SÉQUARD (*Archives de Physiologie*, 1889, 1 and 2, pp. 219-246).

39. Nerve-tracts degenerating secondarily to lesions of the Cortex Cerebri. SHERRINGTON (*Journal of Physiology*, x. 5, pp. 429-432, *July*, 1889).

Preliminary account of the results of observations made with a view of determining the exact number of fibres degenerating in the pyramidal tracts after removal of various portions of the cerebral cortex, their situation and extension in the cord. S. finds that when arm centre only is injured degenerated fibres are found even throughout the sacral regions of the cord. After injury to leg centre many degenerated fibres stop short at cervical swelling. Degenerated fibres are found in both lateral tracts—those on the same side as the lesion being fibres which have "re-crossed" in the neighbourhood of the cervical and lumbar enlargements. The number of degenerated fibres may increase from above downwards. This is due to division of fibres. Two fibres lying close together or "geminal fibres" being found in the crossed lateral pyramidal tract, and very much less frequently in the re-crossed tract. Fibres are not definitely grouped

in the tracts according to the cortical area from which they come.

The fornix-fibres degenerate from behind forwards. Cortical lesion produces degeneration in the corpus callosum, the fibres do not unite identical areas but tend to scatter.

40. Appendix to papers on descending degeneration following lesions in the Gyrus Marginalis and Gyrus Fornicatus in Monkeys. FRANCE (*Proc. Roy. Soc.*, 1889, p. 122).

Tends to establish a differentiation in the pyramidal tract.

CEREBELLUM: HISTOGENY.

41. Histogenesis of Cerebellar Cortex. BELLONCI and STEFANI (*Archives ital. de Biologie*, xi., p. 21, *Jan.* 1889).

Deals with origin of molecular layer and relation to its growth of the external nuclear layer of embryonic brains. The latter is regarded as a germinal layer similar to the malpighian layer of the skin. From its cells originate the radial fibres of the molecular layer.

CEREBELLUM.

42. Atrophy of Fibres in the Cortex Cerebelli. MEYER (*Archiv. f. Psychiatrie*, xxi. i., pp. 197-222, pl. vii., *August*, 1889).

Observation of the medullated fibres in the cortex cerebelli in progressive paralysis and similar diseases.

The cerebellum suffers in the same kind of way as the cerebrum, especially its frontal lobes, and on the same side. There was but little alteration in the number or appearance of Purkinje's cells.

MEDULLA.

43. Nuclei Arciformes. JELGERSMA (*Centralbt. f. Nervenheilkunde*, No. 7, 1889).

These nuclei atrophy in the same way as the nuclei pontis. Like the nuclei pontis they are undeveloped or badly developed in many idiot brains. T. supposes that the fibres which originate in these nuclei are connected on the one side with the cerebellum through the restiform body, on the other side with the great brain by fibres lying in the pyramidal group. T. regards the nuclei as not belonging to the arciform fibres, but as pontal nuclei which have been displaced caudalwards.

CENTRES: LARYNX.

44. Motor-centres of the Larynx. MASINI (*Archivio ital. de Laryngologia*, viii., p. 45, 1889).

NERVES: CRANIAL. I.

45. The Nose and Jacobson's Organ. BEARD (*Morphological Studies*, iv. *Jena, Gustav Fischer*, 1889. *Reprint from Zoologische Jahrbücher*).

Contains an account of the development and structure of Jacobson's organ in the Ophidia and Lacertilia. Concludes that the organ is a specialised part of the nose of much greater sensitiveness than the rest. Larger part of paper devoted to a consideration of the segmental meaning of nose. B. is strengthened in his conviction that the nose and the ear, quite unlike the eye, belong to the same category of sense organs as those of the lateral line or branchial sense organs. Bibliography of Jacobson's organ.

OPTIC CHIASM.

46. The Development and Course of Medullated fibres in the Optic Chiasm of Man. BERNHEIMER (*Archiv. f. Augenheilkunde*, xx., 1 and 2, pp. 133-179, pl. x. *June*, 1889).

NERVES: CRANIAL. II.

47. Experimental and Pathological Observations upon the Optic Centres and Tracts. MONAKOW (*Archiv. f. Psyschiatrie*, xx., 3, pp. 714-788, pts. xi., xii., xiii., 1889).

Anatomical study of brain of cats and dogs operated upon by M. and Munk.

NERVES: CRANIAL. II.

48. A New Centre for the Optic Nerve in the Hen. PERLIA (*Archiv. f. Ophthalmologie*, xxxv., 1, pp. 20-24, pl. ii., *April*, 1889).

By Gudden's method a portion of the optic tract is followed in the hen beyond its usual end-station in the mid-brain to a nucleus (the nucleus of the median optic tract) in the lateral part of the roof of the after-brain on the outer side of the trochlear nucleus.

NERVES: CRANIAL. II.

49. Optic-conducting Paths in Human Brain. RICHTER (*Archiv. f. Psychiatrie*, xx., 2, p. 504).

Atrophy of the optic nerves on one or both sides can, when it affects a grown-up person, be followed with the naked eye as far as the pulvinar and external geniculate body, but after it has existed for a year it will not necessarily be seen on macroscopic observation.

CRANIAL NERVES. III.

50. The Upper Nucleus of the Oculomotorius. DARKSCHEWITSCH (*Arch. f. Anat. u. Phys. Anat. abth.*, 1889, i. and ii., pp. 107-117, pl. vii.).

In the *Neurologisches Centralblatt* Darkschewitsch has described, in the human fœtus, a collection of cells just above the oculomotor nucleus. This he termed the "upper" or "small celled" oculomotor nucleus. He finds that this nucleus is in connection with the ventral fibres of the posterior commissure, which are the first to myelinate.

The upper nucleus has, at least, three connections; (1) with the posterior longitudinal bundle; (2) directly with the pineal gland through the ventral part of the posterior commissure; (3) by certain fibres originating in the nucleus it takes part in the formation of the ansa lenticularis. The posterior commissure contains at least two sets of fibres, neither of which belong to the formatio reticularis.

CRANIAL NERVES. VIII.

51. Origin of Auditory Nerve in the Rabbit. BUMM (*Allg. Zeitschr. f. Psychiatrie*, xlv., 5 and 6, p. 568).

CRANIAL NERVES. VIII.

52. Origin and Central Course of the Nervus acusticus in the Rabbit and Cat. BAGINSKY (*Sitz. der K. P. Akad zu Berlin*, xxxiii., p. 635, *June* 27, 1889).

Destruction of the labyrinth in young animals and examination of the brain seven to eight weeks after the operation by Weigert's staining. Cochlea destroyed on right side, atrophy of posterior root, anterior auditory nucleus and tuberculum laterale. Also on this side considerable shrinking of corpus trapezoides and upper olive, while the upper olive of the opposite side was normal. Deiter's nucleus, inner auditory nucleus, corpus restiforme, pons, cerebellum, brachium conjunctivum, posterior longitudinal bundle unchanged. Atrophy of opposite inferior fillet as far as posterior brachium, but no obvious atrophy of posterior quadrigeminal tubercle could be observed. Extensive atrophy of the striae medullares of the same side. Great difficulty in causing atrophy of the anterior root of the auditory nerve by destroying all the ear except the labyrinth. It was accomplished in two rabbits and one kitten. On the same side the inner division of the cerebellar peduncle (*Meynert*) was diminished in size and its scattered cells atrophied. Small nerve cells on the ventral side of Deiter's nucleus were atrophic. Deiter's nucleus not considered to be in connection with the auditory nerve.

Cranial Nerves. X.

53. Minute Anatomy of the Vagus Nerve in Selachians, with Remarks on the Segmental Value of the Cranial Nerves. Shore (*Jour. of Anat. and Phys., April*, 1889, pp. 428-452, pl. xx. and xxi).

Critical account of previous work on the subject. Method—osmic staining. Conclusions: (1) The vagus in the skate presents a small group of cells on the hindermost of the posterior roots equivalent to dorsal root ganglion. A group on each branchial branch—vagrant ganglia of splanchnic motor roots. A second group on each branchial branch—the pre-branchial ganglia. A group in the visceralis. A group in the lateralis. (2) The vagus of Raia does not contain any non-ganglionated somatic motor fibres. It is not, therefore, a complete metameric nerve. (4) It contains the typical element of the so-called sympathetic system; the proximal ganglia are represented by the branchial and visceralis ganglia, the distal by the pre-branchial ganglia. As will be seen from the extract, *Gaskell's* views and nomenclature are adopted.

Cranial Nerves. XII.

54. A rudimentary Dorsal Root with a Ganglion for the Hypoglossal Nerve in Man. Chiarugi (*Boll. R. Accad. dei Fisiocritici di Siena*, vi., ii.; *Arch. ital. de Biologie* xi., iii.).

Small filament with ganglion accompanying the most caudal roots of the hypoglossal in a child of two. The ganglion appeared to belong to filaments of the dorsal roots of the first and second spinal nerves.

Cord—Topography.

55. Relations between the Superficial Origins of the Spinal Nerves from the Spinal Cord, and the Spinous Processes of the Vertebræ. Reid (*Jour. Anat. and Phys., April*, 1889, pp. 341-354, pl. xiii., xiv.).

After describing method adopted for obtaining an orthographic projection, Reid gives results for each root in his specimens. Finding that "the superficial origin of any individual spinal nerve has no fixed and definite relation to the apex of one, or the apices of two spinous processes, or the space intervening between two, but varies considerably," the author gives a table showing highest and lowest point at which root was situated in the specimens examined.

CORD—SULCI.

56. Anterior Intermediate Sulcus of Human Spinal Cord. BERTELLI (*Soc. Toscane des Sci. nat., July* 1, 1888; *Arch. ital. de Biologie* xi. iii, 1889).

CORD—SENSORY TRACTS.

57. Remarks on Edinger's Paper, "The Continuation of the Posterior Spinal Roots to the Brain." AUERBACH (*Anat. Anzeiger, July*, 1889, pp. 407-411).

58. The Continuation of Posterior Spinal Roots to the Brain. EDINGER (*Anat. Anzeiger*, 1889, No. 4).

Results of comparative studies. In the frog posterior root fibres pass ventralwards through the grey matter and cross to the opposite side in front and behind the central canal; they extend cerebralwards most of them in the anterior, but some in the lateral columns. Same crossing in mammals. As is well known *Edinger* looks upon the fillet as composed of centripetal (sensory) fibres from the cord.

CORD—DEGENERATIONS.

59. Secondary Degenerations of the Spinal Cord. TOOTH (*Goulstonian Lectures reported in British Medical Journal*, No. 1,475, pp. 753-759; No. 1,476, pp. 825-833; No. 1,477, pp. 873-878, April, 1889).

CORD—IRRITABILITY.

60. Stimulation with a Needle of the Spinal Cord of Rabbits, BOECK (*Archiv. f. Anat. u. Phys.*, 1889, parts 3 & 4, pp. 238-252).

Spinal cord, either with or without medulla oblongata, was pierced with a fine steel needle or with one pole of an induction apparatus. The results of pricking-stimuli are not explicable on the supposition that the white columns of the spinal cord are made up of bundles of fibres of similar functions. If it were a change in the place pricked would result in an alteration in the groups of muscles which contract. The effects are regarded as due to reflex stimulation, not to direct irritation of motor paths, since the character of the effects depends upon the presence or absence of the brain.

CORD—REFLEX.

61. The Otospinal Reflex Centre and its Situation in the Cervical Cord. GELLÉ (*Annales d. mal de l'oreille*, No. 9, 1888).

Spinal Ganglia.

62. The Relation of Nerve Fibres to Nerve Cells in the Spinal Ganglia. Gad and Joseph (*Archiv. f. Anat. u Phys.*, 1889, iii. and iv., pp. 199-238).

Authors come to the following conclusions:—Most centripetal nerve paths are interrupted in the spinal ganglia in bipolar nerve cells. The functions of these cells is trophic. Every centripetal stimulus must traverse a nerve cell in the spinal ganglion. There is a delay on the average of .036 *sec.* in the passage of a nerve impulse through the ganglion as found by stimulating first peripherally and then centrally to the ganglion, but whether this is due to summation or not remains doubtful. The nerve after being severed from its trophic centre for two days is found completely functionless. The ganglia are abundantly supplied with blood. When a posterior root is cut on the distal side of a ganglion, all the peripheral fibres die while some of the central fibres on either side of the ganglion, the "post septal," also die. If these fibres have their trophic centres towards the periphery it is difficult to account for their death on the distal side of the section. Authors suggest that some fibres need not only connection with their trophic centres, but also the constant passage of impulses to maintain their vitality.

Sympathetic.

63. Anatomy of the Sympathetic in Birds. Marage (*Annales des Sci. Nat.*, vii. 1-2, 1889, pp. 1-72, plates i.-vi.).

Sympathetic receives most of its fibres from the rami communicantes in the thoracic region. The superior cervical ganglion lies in the angle between the ninth and tenth nerves and receives a ganglionated cord which connects it with the thoracic cord. This single trunk lies in the vertebral canal. Three ganglionated nerves come off from the thoracic sympathetic: (1) the great splanchnic, (2 lesser splanchnic, (3) the intestinal nerve, anastomosing above with the splanchnics and vagus, below with the abdominal sympathetic.

Sympathetic.

64.—Further Observations on the Histology and Function of the mammalian Sympathetic Ganglia. White (*Journal of Physiology*, x., 5, pp. 341-358, *July*, 1889).

In a former paper White concluded that the human superior cervical ganglion is an atrophied degenerate organ. Same pig-

mentary changes observed in the fresh state. Splanchnic and other ganglia also observed. Conclusions that (1) in lower mammals and young human beings the collateral ganglia are functionally active, but that in monkeys there are evidences of the commencing loss of this function, which has completely disappeared in the human adult. (2) In man the function of the lateral ganglia is maintained well into adult life, and only begins to disappear in old age.

65. The Effects of Extirpating the Cæliac Plexus. LUSTIG (*Archivio per le Scienze Mediche*, xiii. p. 129, 1889).

This experiment has yielded various results in the hands of different physiologists, such as diabetes insipus, diabetes mellitus, alterations of digestion, atrophy of the pancreas, &c. Some of these effects may be due to peritonitis. *Lustig* reduces this to a minimum by antiseptic precautions. Rabbits and dogs die, as a rule, in two to three weeks suddenly without alarming symptoms. For first day or two sugar usually appeared in the urine, but subsequently disappeared. Then acetone appeared, and later albumin; the urine diminished in amount; hyaline casts, red and white blood corpuscles, and epithelium were present. In two rabbits which survived acetonuria and albuminuria disappeared.

SYMPATHETIC—VASO-MOTOR.

66. Innervation of the Renal Blood Vessels. BRADFORD (*Journal of Physiology*, x., 5, pp. 358-408, pls. xxiii.-xxvi., *July*, 1889).

67. Method for completely Destroying the Cord in Mammals. Application to the analytical study of Vaso-motor Actions. GLEY (*Comptes Rendus de la Soc. de Biol.*, p. 110, *Feb.* 16, 1889).

In a dog under artificial respiration the medulla is cut across between the skull and the atlas. An opening is made into the spinal canal in the lumbar region. A catheter is passed into the upper opening, and the cord washed out of the lower opening in fragments in from twenty to twenty-five minutes without the least bleeding. The influence of local agencies upon the vaso-motor system was then studied.

68. Vaso-motor nerves of the Head. MORAT (*Archives de Physiologie*, 1889, 1 and 2, pp. 196-211).

NERVES—VISCERAL.

69. Centres for the Innervation of the Small Intestines. PAL and BERGGRÜN (*Mediz. Jahrbücher*, viii., 1888, [*issued June*, 1889]).

Centres for inhibition of the small intestine must lie above the thalamus, the fibres passing through the medulla and corpus striatum to the sigmoid gyrus.

SYMPATHETIC—REFLEX.

70. The Presence of Reflex Vasomotor Centres in the Ganglia of the Sympathetic System. ROSCHANSKY (*Centralbl. f. d. Wissensch. Med.* 1889, p. 162).

The cutting of the cervical cord in curarised cats does not completely prevent the elevation of temperature which is produced by stimulating the central stump of a splanchnic nerve, although it diminishes it. Nor is it altogether abolished by destroying the spinal cord below the level of section, the animal being kept alive by transfusion of defibrinated cat's blood. It is not abolished by cutting the lateral chain between ninth and tenth ganglia, but the pressure is greatly lowered by dividing it between the origins of the great and lesser splanchnics.

NERVES—PERIPHERAL.

71. Innervation of the Muscles of the Soft Palate. TURNER (*Jour. Anat. and Phys.* 1889, *July*, pp. 523-532).

NERVES—DISTRIBUTION.

72. The Localization of Skin Sensibility in its relation to the Sensory Roots of the Spinal Cord. BOCCI (*Moleschott's Unters. z. Naturl.*, xiv. 1, p. 19).

In the frog the posterior root of the seventh lumbar nerve supplies the skin covering the triceps femoris, that of the eighth nerve the skin over the peroneus and tibialis, that of the ninth the skin over the gastrocnemius.

ELECTRIC ORGAN.

73. Electrical Organ of Skate. SANDERSON and GOTCH (*Journal of Physiology*, x. 4, pp. 259-277, pl. xxii. *May*, 1889).

CONSCIOUSNESS.

74. Consciousness and its Limits. BECHTEREW (*Kasan*, 1888, in Russian, *Abstr. Neurologisches Centralbl.*, *May* 1, 1889).

PERCEPTION.

75. The Production of a perception of Pain by Summation of Sensory Stimuli. NAUNYN (*Arch. f. exper. Path. und Pharmak.* xxv., iii. and iv. p. 272, 1889).

INHIBITION.

76. Field of Action of Inhibition in Physiology, in Pathogeny and in Therapeutics. BROWN-SÉQUARD (*Archives de Physiologie*, 1889, 1 and 2, pp. 1-24).

REFLEX—KNEE.

77. On the Nature of the Knee-jerk. LOMBARD (*Journal of Physiology*, x., 1 and 2, pp. 122-149, *February*, 1889).

CELLS.

78. Nerve Cells as Centres of Energy. BOCCI (*Moleschott's Unters. z. Naturl.* xiv., 1, p. 16).

HISTOLOGY.

79. Is the Active Condition of the Central Nervous System accompanied by Changes visible with Microscope? KORYBUTT-DASZKICWICZ (*Arch. f. Mikros. Anat.*, xiii., 1, pp. 51-71, *June* 12, 1889).

Two exactly similar frogs chosen. Sciatic plexus of one stimulated for one hour. Spinal cords of the two frogs then prepared in exactly the same way (hardened in corrosive sublimate, &c.). Sections of each cord stained in hæmatoxylin, nigrosin, eosin, and safranin. The nuclei (? of nerve cells) stained some blue, others red. In the stimulated animal the proportion of red nuclei to blue nuclei was much higher than in the control animal.

80. Certain Constituents of Peripheral Medullated Nerve Fibres. JOSEPH (*Sitzungsb. d. K. P. Akad zu Berlin Dec.* 18, 1888).

Hardening in ½% osmic acid two hours. In the electric nerves of the torpedo, the axis cylinder is to the myelin sheath in cross section as 3-5. In Lophius, frog and rabbit the relation was the same. Two different substances differentiated both in the medullary sheath and in the space in which the axis cylinder lies, that in the former being indentical with *Ewald* and *Kühne's* neurokeratin. The axis cylinder scaffolding remains intact after the nerve has been cut for fourteen days and is therefore considered to be non-nervous. Joseph concludes that the axis cylinder contains two substances (in addition to the scaffolding).

81. Commencement of Schwann's Sheath in the Spinal Roots. SPRONCK (*Nederl. Tijdschrift voor Geneesk*, p. 147, 1888).

The first nodes of Ranvier which appear in the roots being placed all at about the same level form a characteristic flat group.

They lie either within or without the cord and make a morphological boundary between the naked cylinders and the peripheral fibres invested with Schwann's sheath. The commencement of the sheath is conical and it is here surrounded by neuroglea cells which can be followed some distance into the root. The first internode is as long as the succeeding ones, but in a few segments the want of regularity has scattered the nodes, so that they are no longer grouped in a plate.

HISTOGENY.

82. The Development of Certain Cerebral and Spinal Nerves. CHIARUGI (*Anat. Anzeiger*, pp. 31-32, *Jan.* 10, 1889).

83. The Development of the White Matter and Extension of Fibres in the Spinal Cord of the Mouse. LENHOSSÉK (*Arch. f. Mikros, Anat.*, 1889, i. pp. 71-125, pls. 6, 7).

A repetition upon the mouse of Flechsig's work upon man. Conclusion that the appearance of the myelin sheath in the central nervous organs of vertebrates, and especially higher vertebrates, follows the same laws as in man.

(1) Gelatinous substance of Rolando reaches to the periphery; it consists of cuticularised (verhornten) epidermal cells without distinction into connective tissue and nervous elements. (2) Nerve cells and fibre network. (3) White substance and calibre of fibres. (4) Anterior roots. (5) Anterior commissure. (6) Anterior columns. (7) Lateral columns. (8) Posterior roots. (9, 10, 11) Posterior columns and posterior commissure. (12) Pyramidal tracts, successively treated in detail.

84. The Neuroblasts and their appearance in the Embryonic Spinal Cord. HIS (*Archiv. f. Anat. u. Phys.*, 1889, iii. and iv., pp. 249-300, pls. xvi.-xix.)

A continuation of the investigations into the origin of the differentiation of the several elements of which the cerebro-spinal axis is composed. The medullary plate consists at first of a single layer of cells. Between the bases of the epithelial cells, protoplasmic or germinal cells are seen. The epithelial cells are changed into "spongioblasts," which give rise to a network or "scaffolding." The interior of the cells exhibits a distinction into a formed filamentous substance and a transparent soft substance. The former enters into the formation of the network which unites neighbouring cells. On the interior of the axis it constitutes the inner limiting membrane; on the outside it collects into a thick, spongy plate, the "velum confine" (if a synonym

for "Randschleier" must be made) bordered by an outer limiting membrane. The first zone which lies next to the inner limiting membrane is the chief situation of the germinal cells. These are distinguishable from the epithelial cells by their form and by the soft character of their protoplasm. Neuroblasts originate from the germinal cells, they elongate and each gives off an axis cylinder process. Ripe neuroblasts are poorer in chromatin than germinal or transitional cells. Some of the neuroblasts send their axis cylinder processes into the anterior roots, others into the anterior commissure and into the longitudinal columns. The epithelial cells and the scaffolding are the first to appear, nerve cells and nerve fibres appear later. The arrangement of the nervous elements depends upon the scaffolding. The velum confine serves as a kind of filter, which allows the fibres to pass out and also serves as a guide to the formation of the longitudinal fibres.

85. Neuroblasts and their Origin in the Embryonic Cord. His (*Separat-abdruck des* xv. *Bandes der Abhandl. der K. Sächs. Gesellsch. d. Wissensch.* No. iv. pp. 313-372, 4 *plates*, *Leipsic*, *Hirzel*, 1889).

Previous authors have occupied themselves with nuclear figures rather than protoplasm. The scaffold-forming cells His names "spongioblasts," the nerve-forming "neuroblasts," and those of the "mitosis region" germ cells (Keimzellen). The latter lie next to the membrana limitans, between the inner nucleus-free segments of the spongioblasts. No difference is observable between the neuroblasts which send their axis cylinder processes peripherally and those of which the processes remain within the axis. The cell body of the neuroblasts is oval and continued at one pole into the axis-cylinder. The fibre-process and the surface of the nucleus are distinctly striped longitudinally. The neuroblasts are collected into groups. They arise in the innermost layer of the spinal tube in the germ cells and extend subsequently into the second layer. Various cells, transitional in form between the germ cells and the neuroblasts, are described. This change in place is supposed to be due to the active alterations in form which they exhibit. The rapid growth of the axis-cylinder in the first instance is due to a redistribution of the protoplasm. Fresh material must be necessary for its subsequent growth. Transition cells are more numerous than spongioblasts. The numerous apparently naked nuclei, His thinks, are fusiform cells cut obliquely. The similarity between the neuroblasts and spermato-

blasts is pointed out. Spermatozoa, however, are regarded as products of the nuclei—axis-cylinders of the protoplasm. Spongioblasts and neroblasts arise out of the originally single-layered epithelium of the medullary tube. The epithelium is distinguishable into a nucleus free zone, and a zone rich in nuclei. The nuclei overlap one another in such a way that the epithelium appears many layered. The spongioblasts are arranged radially in the inner layer, obliquely in the outer. Each cell gives one internal and one external process and certain lateral processes. The inner unite to form the inner-limiting membrane, and have cross-communications. The network may be made of active protoplasm, or may be but stiff threads. The outer processes divide and anastomose, the connections being very numerous in the neighbourhood of the velum confine (if such a term may be used instead of "Randschleier"). This velum is the bed in which white fibres are laid down. It is made up of lamellæ connected by cross plates. At the end of the second month there appear in the thick spongioblastic network, cells, which His regards as predecessors of the Deiter's cells. The same arrangement of medullary elements is found in all classes of vertebrates with slight variations in detail.

Circulation.

86. Circulation of Blood in the Circle of Willis. Corin (*Archives de Biologie* ix., 1, pp. 17-27, *May*, 1889; also *Bulletin de l'Académie Royale de Belgique*, *3rd series*, xiv., 7, 1887).

Experiments to determine whether ligature of the carotids produces a marked lowering of pressure in the circle of Willis. Pressure in the circle usually amounts in the day to 80 or 90 mm. mercury. Pressure never falls to zero even when both vertebrals and both carotids are tied. This explains the common failure of attempts to produce asphyxia in the dog by tying these four vessels. Pressure in the circle was obtained by connecting the peripheal end of one of the carotids with the manometer; when this was done compression of the remaining three vessels produced but a transitory effect upon the pressure. The failure of this experiment to produce asphyxia is not therefore to be used as an argument against *Rosenthal's* chemical theory of respiration. Manometer in peripheral end of carotid shows no dichrotic movement. The pulse wave is as much retarded as on its journey to the femoral.

CIRCULATION—SINUSES.

87. Experimental Obliteration of the Sinuses of the Dura Mater. FERRARI (*Arch. ital. de Biologie* xi., 1, p. 171, *Jan.* 1889).

CIRCULATION—LYMPH.

88. Cerebro-spinal Fluid. HALLIBURTON (*Journal of Physiology* x., 4, pp. 233-253, *May*, 1889).

CIRCULATION.

89. The Cerebral Circulation in Man in the Normal State and under the Influence of Hypnotic Substances. RUMMO and FERRANNINI (*Resumé in Arch. ital. de Biologie* xi., 2, pp. 272-338, *March*, 1889. 4to, pp. 69, 16 *lithographs* and 17 *engravings*, *Terani*, *Naples*).

Advantage taken of cases of deficiency of the skull. Movements of brain transmitted to a tambour. Variations due to time and meals. During sleep three phases distinguished—(1) From ten to one a.m. ischaemia with constriction of vessels and diminished frequence of pulse; (2) one to three, slower pulse dilated vessels; (3) three to five, increasing vaso-constriction with more frequent pulse. Plethysmographic observations of variations in circulation in limbs, accompanying the brain changes. Similar observations under chloral, paraldehyde, morphine, narceine, hypnone, methylal, and urethane. Some substances, such as narceine and morphine, modify rather the excitability of the nerve centres; others, such as hydrate of choral, paraldehyde and ethyl urethane the cerebral circulation; others, hypnone and methylal, for example, modify the one and the other almost like physiological sleep.

CORD—NUTRITION.

90. Experiments upon the relation of the Motor Ganglion Cells of the Medulla Spinalis to Peripheral Nerves. SARS (*Virchow's Archiv.* vii., 2, p. 243).

NUTRITION.

91. Congenital Neuropathic Papilloma. WHERRY (*Practitioner*, May, 1889, p. 357).

Quoted in this place as distinct instance of nervous control of nutrition. Girl æt. 15. Two patches of dull red rough warty

looking papules, itching intensely. One patch corresponded to external branch of radial, the other to cutaneous branch of ilio-inguinal. The first cured by dividing the nerve, the second treated with potassa fusa recurred.

Nerves: Irritability.

92. Action of heat on muscle and nerve fibres. Moriggia (*Archives ital. de Biol.* xi., 3, pp. 379-389, *June*, 1889).

Frogs plunged in baths of hot water or oil. Nerves retain their irritability at temperatures at which muscles coagulate.

Heat.

93. Thermopolypnoeic centre and thermotaxis. Ott (*Phil. Med. News*, 1889, *No.* 4, p. 109).

Microcephalus.

94. Description of three microcephalus brains, and anatomical study of microcephalus. Part I. Marchand (*Nova Acta der k. Leop-Carol. Akad. d. Naturforscher*, Bd. L. iii., iv. 3, 5 plates, 1889).

Microcephalus.

95. Encephalon and Skull of a Microcephalus. Mingazzini and Ferraresi (*Moleschott's Untersuchungen z. Natur. d. Menschen u. d. Thiere* xiv., 1, p. 103).

Method.

96. A Modification of Weigert's Method for coloring the nerve centres. Vassale (*Riv. speriment. di Freniatria, &c.*, xv., p. 102, 1889).

Tissue hardened in Müller's fluid or bichromate of potassium. Slight alteration in fluid as used by Weigert.

A. Hæmatoxylin 1 gramme, hot distilled water 100 gr.

B. Saturated filtered solution of neutral copper acetate.

C. Borax 2 gr., potassic ferrid-cyanide 2·5 gr. in 300 gr. water. Sections lie 3-5 minutes in A, and an equal time in B. They are quickly washed in C and thus decolored, dehydrated in absolute alcohol, cleared in 1 part carbolic acid to 3 parts xylol and mounted in xylol balsam.